

LECTURES
ON
THE PHYSIOLOGY AND DISEASES
OF
THE CHEST,
INCLUDING
THE PRINCIPLES
OF
PHYSICAL AND GENERAL DIAGNOSIS.

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TO THE

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LECTURES
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LECTURE I.

Introductory remarks on the claims of diseases of the chest to be separately considered—Importance of studying their physical as well as their general signs—Illustrations—Practical insufficiency of either class of signs separately—Advantages of studying both rationally—Analysis of the mechanism of respiration—Forces and mechanism concerned in dilating the chest, upwards, outwards, and downwards—Forces which diminish the cavity of the chest—Modifications.

GENTLEMEN,—A course of lectures devoted specially to the consideration of the viscera of the chest in their healthy and diseased state, may be open to the objection, that any partial study of a set of organs and their diseases tends to engross the mind with a disproportionate attention to these organs, to the neglect of general disorder of the system, and of affections of other important viscera. In answer to this objection, we might urge the great prevalence and fatality of thoracic diseases in this country; constituting nearly one *half* of the fatal cases, and perhaps quite one *half* of the slighter disorders; and we might strengthen this argument by the fact, that many other diseases, both local and general, owe their serious or fatal character to secondary thoracic lesions. In fact, such is the immediate relation between the thoracic organs and life, that when diseases of other parts prove fatal, it is by arresting the functions of those organs, and when the process of this arrest is slow, morbid conditions are often produced in the lungs and heart, resembling those of their primary diseases, which speedily accelerate the fatal event by their superadded influence. It is thus found that in many of those who die of lesions of the brain, abdominal viscera, &c., especially those of a chronic character, the lungs are congested, and even hepatized, and effusions of serum and of lymph are found in the thoracic cavities.

But the great prevalence and fatality of thoracic diseases are not sufficient reasons for making the lungs and heart exclusive objects of study; the condition of extra-thoracic organs in these as well as in other disorders requires notice; and it will be a leading aim of

the following lectures to direct attention continually to the general states of the system, as well as to the local disorder, and to show how important it is for the practitioner to instruct and guide himself by all the symptoms, general and local, vital and physical, in combination. I would, therefore, rather give another character of thoracic diseases, as a sufficient reason for treating of them separately. They are, in the present state of our knowledge, more than any other internal diseases, susceptible of clear and conclusive illustration, and this for two reasons:—First, because the structure and functions of the organs within the chest are, for the most part, simple and intelligible; secondly, because these organs, in the performance of their functions, have certain mechanical and physical relations, which may bring their conditions, healthy and morbid, under the more or less immediate cognizance of our external senses; so that our study of the most serious internal affections may be approximated to that of simple external disease.

Studied merely through their general or animal signs, thoracic diseases are often as equivocal in their character and insidious in their progress, as they are serious in their tendency. Thus, *pain* is often absent in affections of the worst character, whilst it may be acute in trivial complaints. *Cough* depends on an undue irritation, direct or by sympathy, of the lining membrane of the air-tubes, a part only of the respiratory machine, and is by no means proportioned to, or determined by, the extent of the thoracic disease. *Dyspnœa* also depends on the sensibility of the nerves with regard to the change of the blood which should take place in the lungs, which sensibility we know to be influenced by a variety of causes foreign to the lungs themselves. The *pulse*, that “*res fallacissima*,” is often not proportionately influenced by a visceral lesion, or when it is so, does not indicate the seat or extent of that lesion. So also *fever* is an affection of the whole system, and is neither a measure nor a constant concomitant of the maladies of individual organs. Thus we may have inflammation of the lungs, nay even of the pleura, without pain, without dyspnœa, with little or no cough, without any constant character of pulse, or degree of fever. We may have cough, severe pain of the chest, dyspnœa, strong and quick pulse, and febrile disturbance, without inflammation of the lungs.

I will give you an example, which is not a very rare one:—An intimate friend of mine, a physician in the country, who was distinguished no less for professional abilities, than for a highly conscientious candour, was called to see a gentleman of middle age, who had long been suffering from dyspepsia, and who appeared to my friend to have one of the attacks of his usual complaint. There was no other pain than what he had been often accustomed to in his common ailment; he felt no dyspnœa, but only temporary oppression, as often before, from flatus; he had no cough; his pulse did not exceed 80; and there were nausea, and a feeling of general uneasiness, but no more fever than what frequently accompanies

what is called a bilious attack. Such the complaint was considered to be, and as such it was treated. After several days of no remarkable change in the symptoms, the physician was suddenly called to the patient, whom he was shocked to find pale, cadaverous, bathed in a cold sweat, with an intermitting, thready pulse, and obviously dying. The change, or *sinking*, came on only six or seven hours before death; and yet, on inspection, almost the whole of one lung was found in an advanced stage of hepatization; a sufficient proof that extensive inflammation must have been carried on for several days, although it gave no indication of its presence by general signs.

I will give you an example of a still commoner case, in which, if the general signs were less negative, they were still insufficient to attract the attention of the medical attendant. A practitioner asked me to see a patient, whom he had been treating for a *liver affection*. It was a labouring man, and he complained of great weakness, with some pain low down on the right side, which was full and tender in the region of the liver. The skin was slightly jaundiced, and he had profuse perspiration every night. The complaint began gradually, and obliged him to leave off work, he said, through weakness, which was increasing in spite of the tonic remedies which he then used. Although I was fresh from Paris, and the wards of Laennec, I was almost ready to fall into the opinion of the surgeon; but finding, on more minute inquiry, that there was a slight cough, I began to examine the chest. One tap of my fingers was enough to reveal where the chief disease lay. One side of the chest, the right, was full of water, the effusion consequent on a latent pleurisy. This extensive local disease, although not evident by distinctive general signs, had already begun to oppress the functions and reduce the strength, and if still neglected, it would most probably soon have taken on a chronic form, accompanied by changes of structure, over which medicine exerts very little control, and if then discovered, the body would have been wasted and worn down before any beneficial influence could have reached the real seat of the lesion. As it was, a series of large blisters to that side, mercurial diuretics, followed by tonics and similar appropriate treatment, soon effected a cure.

Such cases as those now mentioned are by no means very rare; many examples have happened within my own observation, where a latent pneumonia or pleurisy has been revealed only by the scalpel after death. In many instances, too, the same lesions have been discovered during life as unexpectedly by auscultation; and if the discovery has not been always followed by a successful treatment, it has at least saved the practitioner from errors which would have degraded his skill in the eyes of others—errors with which his conscience would have been often haunted in hours of despondency and self-distrust. But in many cases, such a discovery of the true nature of the disease has prompted a line of treatment which has proved palpably successful; and the certainty of the information thus obtained gives to the practice a decision and an energy that a

conscientious man can never bring to bear in cases of perplexity and doubt.

It has occurred to me in a good many instances, to see patients supposed to labour under complaints called bilious, dyspeptic, nervous, or designated by the more convenient term general debility; and these patients do truly exhibit many symptoms of biliousness or dyspepsia, nervous irritability or weakness: but instead of being benefited by the remedies usually resorted to in such affections, calomel and black dose upset them, peptic remedies do them no good, active exercise aggravates all their complaints; tonics and stimulants are equally prejudicial; and the patients have gone from doctor to doctor, getting nothing better than temporary relief, and oftentimes getting something worse. In these cases, the liver, the stomach, the nervous system, the general strength, have truly suffered, but they have suffered secondarily. A disease, either functional or structural, of another organ, has been the true *fons et origo mali*; and that organ is the *heart*. This, by failing in its important duty of carrying on the circulation, that spring of all functions, whether its propulsive force be insufficient, or whether its valvular apparatus admit regurgitation, involves other organs in disorder; viscera are congested, secretions depraved, sensibility deranged, functions disturbed, muscular power impaired; and all these effects may be obvious and distressing, whilst the original cause is either imperceptible or eclipsed amid the crowd of mischief that it has raised. Now I do not mean to assert that in all such cases physical signs will discover the cause, or by discovering it will enable us to remove it; but I can state with some confidence that they will often discover it, and they will then generally enable us to direct our practice more successfully and more safely than we could do without them.

I might easily multiply illustrations, but let one more suffice. The late epidemic, so injudiciously designated by the single name influenza, in many instances comprehended the most serious inflammations of the thoracic viscera, which were masked by the general symptoms, and were to be detected among the catarrhal and bilious affections common to the disease, only by means of the physical signs.

For the sake of my junior hearers, it may be well to explain here that *physical signs* are those which the diseased part can impress on our external senses, directly or through some physical medium. Thus the ocular examination of an external disease—the feeling of a tumour within the reach of the fingers—the listening to sounds depending on the form, position, or density of internal parts—all furnish us with *physical signs*; which, as they are produced by invariable and intelligible physical laws, are free from the fallacies of general symptoms, which depend on the varying and more mysterious properties of animal life—such as sensation, irritability, and sympathy.

While I would thus maintain the importance of attention to

physical signs, I am very far from wishing to supersede the study of constitutional conditions. Let physical signs be taken according to their worth. They inform us of local lesions of structure or of function; but they do not tell us of the relation of those lesions to the body at large, or to other parts of the body: diseased function and condition may be in other parts also; and whether as cause or as effect of the local physical lesion, they are as much as this to be considered in estimating the condition of the patient and in determining the method of treatment. Thus, in the typhoid form of inflammation of the lung, the physical signs scarcely indicate any difference in the condition of the lung from that in common inflammation; but the general signs—the weak thready pulse, the clammy surface, the brown tongue, the oppressed senses, and the extreme general prostration—these show a state of the body of far more importance than the local disease as declared by the physical signs, and in a more imperative degree requiring its treatment.

Again, some morbid conditions which give physical signs are not enough characterized by these alone; but when their constitutional symptoms are also considered, the physical signs then appear in their true light. Thus œdema of the lungs has the same physical signs as the first stage of pneumonia; but the absence of fever, of pain, and the presence of anasarca in other parts, will generally supply what is wanting to distinguish the former from the latter affection.

Further, the positive indications of physical signs, although in themselves accurately detecting disease of an organ, will often be insufficient alone to guide the treatment. Thus organic valvular disease of the heart I may detect by physical signs alone, and I may through them specify with exactitude its nature and extent, but the course of treatment will depend on the general state, the bodily strength, the vascular fulness, the degree of nervous excitability, and the condition of other functions; whilst, at the same time, the organic disease of the heart which the physical signs alone may have discovered, must also be held in view as a permanent influence to be duly respected in the whole course of the treatment.

Even thus limited, the importance of physical signs will still appear to predominate; for if positive in character, they tell us far more of the real nature and amount of the organic disease than any assemblage of general symptoms; they are more truly the representative of the lesions themselves; in fact they are absolutely part and parcel of these lesions. To illustrate this, a patient with peripneumonia may have severe pain, little pain, or no pain at all—much or little cough; he may be oppressed by dyspnœa, or not be sensible of it at all; his pulse may be soft or hard, and variously quick; there may be thirst, anorexia, heat of skin, and other indications of fever, or they may be scarcely present; and these several symptoms are so far from marking the disease, or bearing proportion to its extent, that they may all exist in a high degree without pneumonia, and pneumonia may be present without enough of these

symptoms to attract attention. But pneumonia cannot exist without changing the physical condition of the lung—the physical condition of the lung cannot be changed without an alteration of its physical properties—and these physical properties may, in the majority of cases, be made more or less evident to our senses. We study these physical properties by the senses that can reach them; we watch and feel the motions of the respiratory machine; we listen to the sounds which they produce, and to the sounds which we can excite in them. When we detect in these movements and sounds those modifications that experience and reason have shown to depend on inflammation and condensation of the pulmonary tissue, we know, almost with the certainty of seeing, that pneumonia is present; and the extent of the signs thus shown, will be a pretty accurate measure of the extent of the lesion. On the other hand, although there be many general signs which seem to denote the existence of peripneumony, if, on careful examination, none of the physical signs are found, we may well distrust the affirmative general symptoms with regard to the diagnosis, although, if positive, they are not to be disregarded in the general treatment. Both sets of signs have their value; and it will be my especial object, in the course of these lectures, to point out modes of appreciating each in the study and treatment of the diseases—to show as much as possible what they are severally worth, by examining them more fundamentally than has generally been done—and, whilst we pay due respect to the records of experience, in whatever shape they may come before us, to make them still more profitable and instructive by careful analysis and moderate generalization.

Now, then, what must be our course to attain this knowledge that shall be at once rational and available? We must imitate the practical philosopher, who wishes to use, repair, or improve a complicated piece of machinery: he studies the springs, the wheels, and all the parts of his machine, their uses and relations to each other, and the laws according to which they act severally and in unison. We must likewise study our machine; we must duly consider its construction—the form, position, and connexion of its several parts; their relations to physical laws, and the combinations of these relations in the rest and in the working of the whole. But in doing this, we shall soon discover that the object of our study is more than a mere machine; we shall find springs in it that are not elastic—chains that are not mechanical—phenomena present themselves which imply properties other than those of mere matter. The laws which regulate these properties beyond those of mere physics, claim also to be carefully studied. We have the *vital* properties, sensibility, irritability, contractility, added to the mechanism—we have a vital chemistry pervading the materials. Besides the chest, which is mechanically enlarged and diminished, and the bag of the lungs and their tubes, which are in like manner expanded and compressed, and the heart and its hydraulic pipes, through which a liquid is propelled, there are in these several parts

also the properties of feeling and sympathy, of irritability and the power of secretion; and these not only bind together, in new and complicated relations, the various parts and organs of the chest, but they connect these severally no less with the other organs and members of the body; and the affections resulting from these varied relations, must have their proper share of our attention.

Thus, then, again arises the division of signs into physical and general; the one class confined to the organs and their physical properties, as manifested in these organs; of the other, although a few may be located in the parts, many, through the various vital properties that link its parts together, are expanded over the whole frame; and thus becomes further evident the necessity of duly appreciating the study of both classes of phenomena in the investigation and treatment of disease.

With these objects in view, we proceed, then, to examine briefly the general structure of the chest and its organs, with such a concise account of their functions as may be necessary to prepare us more fully to understand their diseases. We shall then be able to study the relations of these organs to physical laws, by which their condition may be examined, and the physical signs of health or of disease may be detected. As we enter this subject, it may be necessary to illustrate familiarly a few points of a branch of physical science, acoustics, which is by no means generally understood, and which is yet quite necessary to the right comprehension and appreciation of the phenomena of auscultation.

Now you see that we have chalked out for ourselves no trifling undertaking; and for me to succeed in it, and for you to profit by it, continued attention on your part will be as necessary as in any other study which is progressive and systematic. I have been asked by several, "When do you begin your lectures on the stethoscope? we want to know how to use the stethoscope." Why, gentlemen, the stethoscope stands in the same relation to this course of lectures, as that in which the telescope does to astronomy. To know how to use the stethoscope is as soon learnt as to know how to look through the telescope; but unless your only end be curiosity or wonder, you will use these instruments to no purpose, without studying, and understanding also, the phenomena which they reveal, and the laws which govern these phenomena.

It is only by this fundamental mode of study that we can attain any thing like a complete or really safe knowledge of the physical signs of disease. To burden the memory with a long and unintelligible list of dogmas, that such and such phenomena indicate the presence of such and such lesions, without endeavouring to instruct the understanding as to the connexion between them, will be as useless and fallacious in practice, as it is unworthy of a branch of science.

The chief advantage of physical signs is in their being *intelligible* and *explicable*; that is, referable to known laws or classes of phenomena. To understand these laws and their fulfilment in the

various phenomena of matter, is the object of natural philosophy ; and if we expect physical signs to aid us in the study of disease, we must examine them by those modes in which simpler nature and her signs alone become intelligible, in their relation to fixed principles and simple laws. The Hippocratic physician, who studies only general signs, must often content himself with registering them in his memory, because many of these signs depend on vital properties which are little known, and occur according to laws which have been but imperfectly developed ; but to extend this uncertainty and imperfection to *physical signs*, the phenomena of *known laws*, the results of well-defined properties, is to carry into physics the doubt and mystery of medicine, and to deprive pathology of the only advantages of certainty and simplicity which physical science is capable of bringing to it. Vital properties, moreover, also have their laws ; and the more we can keep these in view in the study of general signs, the more useful and instructive will be the information which we derive from them.

We have not time to enter into a minute detail of the anatomy of the chest, nor, indeed, is it necessary. I presume that you are already familiar with its structure, and I have now only to advert to the chief constituents of the machinery of respiration, and the mode in which these work.

The machine of respiration was compared by Mayow to a bag within a bellows, the bag alone communicating with the external air, and becoming full or empty as the bellows expands or closes. The comparison must be admitted as only a rough one, for there are many points of material difference. The alternate enlargement and diminution of the chest are certainly the chief movements in respiration ; and in proportion as these are complete, and those of the lungs harmonize with them, the mechanical part of respiration will be effectually performed.

Now the chest is, as you know, enlarged essentially by muscular action, but its diminution is chiefly effected by the elasticity and weight of the parts, occasioning their collapse or subsidence from that condition to which the muscular action had brought them. Thus in ordinary breathing, inspiration is active, and expiration in a great measure passive, and the period of action is alternated with a much longer period of rest. As these motions of the respiratory machine constitute an element important in the production of many signs, we may well bestow a few more minutes upon them, especially as they do not seem to be fully understood by some modern pathologists.

Before we describe the means by which these motions are effected, let us illustrate their nature by those of the organs on which they operate. Here are healthy lungs, and observe the character of their expansion when I inflate them : their summits rise, their middle portions expand, and their bases and margins descend and spread. Their motion is, therefore, peripheral, or radiating from a centre : now where is this centre or most fixed point ? Why, not at their summits,

as some modern writers have erroneously supposed, but at their roots, where they grow, as it were, from the bifurcated trunk of the trachea, which, where it again divides, becomes in a measure fixed by its attachment to the great blood-vessels and the spine. So also shall we find the enlargement of the chest to be peripheral nearly in all directions, the spine being the fixed centre. Its chief increase is indeed downwards and around its lower portions; but if we watch the upper parts with relation to the spine, we can plainly see them also rise and expand considerably. This general enlargement of the chest may be analysed into its enlargement *downwards, upwards, and outwards*.

The enlargement of the chest *downwards*, is, as you know, effected by the action of the diaphragm, which in its passive state projects in a very convex form upwards into the chest, but when acting, its muscular margins and pillars draw its tendinous centre downwards towards their attachments to the lower margins of the ribs and sternum, and the upper lumbar vertebræ. In the same contraction the muscular portions of the diaphragm being straightened towards the centre, touch the ribs at fewer points, and form angular spaces, in which the thin marginal lobes of the lungs become expanded downwards. Now as this downward action of the diaphragm cannot take place without pressing on the contents of the abdomen, it causes an outward movement of these contents, and the swelling of the abdomen at each inspiration therefore becomes an index of this action.

The enlargement of the chest *upwards*, by which the apices of the lungs are expanded, is effected by the drawing upwards of the upper ribs, sternum, clavicles, and scapulæ, by the sterno-mastoid, scaleni, and other muscles, which connect this upper framework of the chest directly or indirectly with the upper end of the spinal column. This motion, although not great in ordinary respiration, is yet in proportion to the extent of the upper lobes of the lungs. It has been overlooked by many physiologists, but it is highly important as a source of diagnosis; and you may perceive and measure it if you keep steady the spinal column by standing with the back against a pole, or the edge of a door, or the corner of a wall; you will then see that the upper ribs rise to the amount of half an inch or more in each inspiration; and so far from being, as Haller and others maintained, almost fixed, they are, in proportion to their length, actually more moveable than the lower ribs. If you examine their attachments to the vertebræ, you will there perceive that the upper ribs admit of more motion than the lower. Magendie, I believe, first pointed out this important part of the movements of respiration, and you now see how it accords with the expansion of the lung which I have just exhibited to you.

The *outward* enlargement of the chest is mainly effected by the contraction of the intercostal muscles. I dare say that you may have heard or read of the long discussions which were formerly excited by this apparent paradox, that of a cavity *being enlarged*

by the *contraction* of its walls. It is, indeed, a mechanical process hard to be described, and yet how plain and simple it is on inspection ! See how these ribs, especially the lower, in their collapsed state, are convex downwards in the portions anterior to their centres. Well, now, these are their most moveable portions, and if they are drawn upwards, the ends being comparatively fixed, you see that their downward convexity is diminished, and their outward convexity or inward concavity is enlarged, and thus the horizontal diameter of the chest is increased.

But observe, for this effect it is necessary that the ribs should be drawn *upwards* ; and what is it that makes the contraction of the intercostal muscles draw the ribs upwards rather than downwards ? Haller and others say it is because the upper ribs are fixed, and cannot be drawn downwards ; but we have just seen that they are not fixed : the fact is that they are drawn upwards themselves by muscular action, and this upward traction is communicated and increased to the ribs below by the contraction of their respective intercostal muscles. If instead of drawing up, or fixing the upper ribs, you draw down, or fix the lower ones by the abdominal muscles, then the intercostal muscles become means of diminishing the diameter of the chest by drawing the ribs downwards ; and they actually do contribute to this end in forcible acts of expiration. Now here is a case illustrating the necessity of learning the mechanism of the chest rationally, and not merely by rote. You see that both sets of intercostal muscles, external and internal, must tend to elevate the ribs as long as the upper ribs are either fixed or drawn upwards ; but when the lower ones are fixed, the contraction of the intercostals must approach them to this lower fixed point.

Well, now, let us go back a moment, and apply the same question to the diaphragm, whose *ordinary* action we found to be to enlarge the chest downwards by the drawing down of its central convexity. This supposes its tendinous centre to be the most moveable attachment of the muscular portion of the diaphragm, that to the lower margin of the ribs and sternum being more fixed. But the centre of the diaphragm is sometimes more or less fixed, and prevented from descending, by tumours or excessive tenderness of some of the abdominal viscera below it. How will its muscular margin then act ? Why, it will draw upwards its lower attachment with the ribs and sternum, and thus increase the capacity of the chest by raising it from the fixed convexity of the diaphragm. When, therefore, you see a patient breathing merely by the heaving of his chest, you are not to suppose that the diaphragm is inactive, for it may thus contribute to inspiration that is wholly *thoracic*. This upward action of the diaphragm is not considerable in common cases ; but it must always be something—that something being the amount of resistance offered to the descent of the diaphragm by the contents and walls of the abdomen.

There is another particular in the ordinary action of the diaphragm which is worthy of your notice, because it may prove a

source of physical signs. You see, by these diagrams, that considerable parts of the upper abdominal viscera, the liver, the stomach, and the spleen, although below the diaphragm, yet are contained within the walls of the chest. Now the portions of these walls which contain them differ from those above the diaphragm, in being subjected to the ever-repeated outward pressure of these viscera, pushed by the descending diaphragm; and the result of this pressure is a permanent bulging or prominence in the lower part of the chest, and a slight furrow or depression above it. This depression generally marks the situation where the lung begins, and where the abdominal viscera cease to be in contact with the walls, although they generally rise above it towards the centre of the chest. The existence of this furrow, and its use as a sign of limits, were first pointed out to me by my friend Dr. Edwin Harrison, and I think I may explain its production by the outward pressure of the sub-diaphragmatic viscera. The position of this furrow varies in different subjects, but may generally be traced from the lower end of the sternum running horizontally around the chest, about, but not parallel with, the seventh and eighth ribs at the sides.

The effect of the reiterated outward pressure of the sub-diaphragmatic viscera is remarkably seen in the eversion of the lower ribs and sternum in rickety children, the bones and cartilages being permanently bent by it. If the belly in these cases be also tumid, the upward action of the diaphragm will draw the sternum forwards and upwards, forming what is called a *chicken-breast*.

We need not dwell long on the ordinary means by which the capacity of the chest is diminished. When the diaphragm ceases to contract, the weight of the viscera and walls of the abdomen force back the diaphragm to its wonted vaulted projection into the thorax: the same weight to the lower ribs, together with the elastic torsion of their cartilages and ligaments, make the ribs collapse on the relaxation of the intercostals and the muscles which raise the upper part of the chest. The merely mechanical constitution of the chest is in favour of its diminution, and so is that of the lungs, which we shall notice by and by; but expiration may be most powerfully assisted and increased by a great many muscles, especially the abdominal muscles, and all those connecting any of the ribs with a part of the spine *below* the attachments of these ribs.

So, likewise, forcible inspirations are assisted by the action of all those muscles connecting any of the ribs with any part of the spine *above* the attachments of these ribs.

LECTURE II.

Internal mechanism of respiration—Form of the air-tubes and cells—Structure of the air-tubes—Cartilages—Mucous membrane—Longitudinal fibres—Circular fibres—Connecting and investing tissues of the lung—Blood-vessels of the lung—Chemistry of respiration—Changes of the air and of the blood—Physical cause of the change of colour of the blood—Adaptation of mechanism to chemical action—Ciliary motions of the bronchial mucous membrane.

BUT now let us proceed to examine the internal parts, to the functions of which the exterior motions of respiration, which we have been considering, are subservient.

Unless the air enter and pervade the lungs freely each time the chest is enlarged, and unless it find as ready egress from them as the chest is contracted, the external machinery, however perfect it may be, will work in vain. Now if we examine the structure of the lungs and their connected tubes, we shall find great cause to admire the beautiful provisions displayed in every part to prevent this want of harmony, and to facilitate and regulate the passage of air through them ; and we shall meet with further illustrations of the nature of these provisions in the modifications which disease can produce in them.

The lungs may be described as essentially constituted by the multiplied divisions of the expansible air-tubes ; and we shall obtain the best knowledge of their structure by tracing these tubes from the trachea to their terminations.

You must all be sufficiently familiar with the form and appearance of the windpipe and its larger branches. Their form and open calibre are given to them by their cartilages, which, in the trachea and the two first divisions, are in rings, almost surrounding the tube—in the next order of bronchi, constituting irregular pieces ; but at the origin of each branch still nearly annular, to keep open the orifice. These plates are fewer as the bronchi, on subdividing, become more slender, those at the orifices of branches being only half rings : and they cease altogether in the tubes of half a line in diameter, the tubes then being membranous to their terminations.

What are these terminations ? Malpighi supposed the lungs to consist of a series of vesicles, freely communicating like those of sponge, and that in these the minuter bronchi terminate. Helvetius conceived that the vesicles of the lungs were only common cellular texture, filled with air, and forming spongy sheaths around the pulmonary blood-vessels, and freely communicating with each other. Haller, judging from the simple vesicular lungs of frogs, &c. came to nearly the same opinion. Even lately Magendie held the notion that the vesicular structure of each lobule was common, having free communication between its cells, besides through the bronchial tubes distributed through it ; and a similar view seems to be entertained by Bourgerj.

The researches of Reisseissen are the most complete and satisfac-

tory of any that have been made, and I shall avail myself of some of his descriptions to convey to you an idea of the minute anatomy of the lungs.

By various modes of injecting, he showed that the bronchial tubes end in *culs de sac*, without mutual communication, except through the bronchi, and without communication with the interstitial cellular texture. Thus by inflating a minute bronchus, the cells of the portion of lung supplied by it became distended as regular roundish vesicles; and, by tying the bronchus, remained so, without any air passing to the adjoining cells. Mercury injected into a single bronchus gave the same result.

If, on the other hand, the common cellular texture be inflated through a minute cut in the pleura, the air is diffused in angular and irregularly-sized bubbles, giving inequality to the pleural surface, and appearing, as in the manner of a common emphysema, more between than in the lobules. Mercury poured into a bronchial tube, and allowed to run to its extremities, is seen in little globules under the pleura, communicating by branches at first very numerous and small, which again run into fewer and larger. If this be pressed between two glasses under the microscope, the mercury then goes to the extremities, and takes the regular cauliflower shape of the extreme canals.

Another mode adopted by Reisseissen to show the shape of the minute extremities was this:—A portion of lung was kept some days in water, so that only very little air remained; hot water was then poured on it, which distended this air, and displayed the expansions of the terminal tubes like little bunches of buds or berries. [These appearances were exhibited in drawings.] I have repeated and verified these observations, and I think we may admit as correct Reisseissen's description of the form of the terminal bronchi.

Now, then, having considered the form, if we examine more minutely the component structures of the bronchial tree, from its trunk to these bud-like termini, we shall find that each is worthy of notice, and that there are some remarkable differences in the several parts. Thus we have seen that the cartilages—large and almost annular in the larger tubes, forming smaller and irregular segments in those of middle size, and ceasing almost entirely in the inner tubes—answer the important purpose of holding open the tubes in those parts where they are apt to be exposed to pressure. If the tubes were merely membranous, they might collapse together by atmospheric pressure, or by the distension of the pulmonary tissue at the upper parts of the lung, and be prevented from transmitting air to their extremities, whilst the absence of cartilage in the smaller tubes permits their flexibility and distension. Well, besides this cartilaginous frame-work, which varies according to the parts, we have at least three other structures, appearing in every part of the air-tubes.

1. A mucous membrane, with many muciparous crypts or follicles, conspicuous in the larger tubes, some of which penetrate through

the other tunics, even to the cartilaginous coverings. In the smaller tubes this membrane becomes fine, thin, and destitute of distinct follicles, and in the terminal cells is of the greatest tenuity.

2. Longitudinal fibres, which are very elastic, like the elastic coat of the arteries. They are very conspicuous in the large and middle sized bronchi, but they can be traced to the very ends of the tubes ; it is these chiefly which cause the lungs to contract and collapse, when the chest is opened after death ; and to a certain extent during life they must therefore assist in the act of expiration. Here you see them in the human bronchi [exhibiting a drawing], and here in those of the calf, in which they are more distinct and numerous than in man.

3. Circular muscular fibres, which are to be found in every part of the bronchial tree. In the larger tubes they are inserted into the ends of the annular cartilages, thus completing the rings. In the smaller they encircle the whole tube, some fibres being attached to the cartilaginous pieces, and to the longitudinal fibres, and some passing over them ; and, by the aid of a lens, they can be seen, especially in the lungs of the lower animals, at the very extremities of the canals. The extreme bronchi have been shown by experiment to contract during life, on the application of a mechanical or chemical irritant ; and we shall soon find reason to suppose that their function renders expiration to a certain degree active. When we come to the pathology of asthma, we shall find other proofs of the irritable contractility of these bronchial muscles, and that their share in the process of respiration will serve to explain many facts which were inexplicable before the existence and action of these muscles were demonstrated. We shall recur to this subject when we come to the vital properties of the respiratory organs. The annexed drawing from the human subject exhibits a distinct view of the longitudinal, and of these circular or transverse fibres.

Thus, then, we have found in the respiratory apparatus a chest capable of enlargement and diminution ; and within it not a mere bag, or assemblage of bags, but a series of elastic, expansible, and contractile tubes, subdividing into innumerable branches, each terminating in blind enlargements, and constituting the chief part of the parenchyma of the lungs. But this congeries of tubes and terminal cells is compacted together by other structures. There are the blood-vessels distributed over them, for the important purpose which we have afterwards to consider ; there is an interstitial cellular texture, uniting the bunches of tubes and their vesicles into lobules, and in a more condensed form uniting these lobules into lobes ; and there is an elastic serous membrane, a part of the bag which lines the whole chest, investing this congeries of structures. This packs them into an organ with a simplicity of outward form which adapts it well to fill the changing capacity of the chest, while the mobility and elasticity of the connecting and investing structures still leave unimpaired the permeability and expansibility of each individual tube. Thus, when one part of the chest expands more

FIG. 1.



FIG. 2.

FIG. 1.—Division of the human Trachea and Large Bronchi, showing the longitudinal and circular fibres. *a* Brachial Glands.

FIG. 2.—Ultimate division of the Bronchi into clusters of air-cells. Magnified three diameters.

than another, the air presses most directly through the tubes leading to that part, and an equality of pressure is thus kept up between the chest and lungs, which is highly favourable to their harmonious working.

Although the elasticity of the bronchi gives the lungs a natural ten-

dency in favour of contraction, so that, when not prevented by the pressure of air within, they collapse, yet the whole lungs will always *admit* of more distension than that which is required of them in respiration; so that if parts become obstructed, the air may enter more freely into other parts, for a short time without inconvenience; but, as we shall see hereafter, the continuance or excess of this partial distension is capable of producing permanent disease of a remarkable character.

Having thus sketched the route and the mechanism by which air passes to and fro in the lungs, the question, why does it so pass? brings us to another anatomical element to be considered—the blood-vessels, which are carefully and minutely distributed around the *air-tree*, especially its finer terminal parts. It is quite unnecessary for me to go here into any common details respecting the general circulation: I conclude *that* to be known; and I would reserve the particulars of the structure of the heart and great vessels, which deserve especial notice, to that part of the course in which their diseases will be considered. Blood of a dark hue is conveyed from the right ventricle of the heart through the pulmonary artery to the lungs, and it returns by the pulmonary veins into the left ventricle, of a florid red colour. Now by what ways does it pass as it undergoes this change? Minute anatomy has supplied us with an answer. It had long been known that fine injections would pass from the pulmonary artery to the pulmonary veins, or the converse, there being no valves in the veins of the lungs; but the actual communication of one with the other, through a set of capillaries on the sides of the minute air-tubes and cells, was first clearly demonstrated by Reisseissen. But it has since been seen in lungs of living frogs and other animals. In these drawings, which are enlarged from those of Reisseissen, you see this communication. In this, again, you see another set of vessels, the bronchial arteries, which are much smaller; they supply the mucous membrane and interlobular tissues, and may be seen here anastomosing with the pulmonary vessels. Branches from both bronchial and pulmonary arteries go to the pleura. The bronchial veins appear chiefly to empty themselves in the pulmonary veins.

The great object, then, of all the apparatus which has been occupying our attention, is that the blood and the air shall be continually moving in close proximity to each other. We have said that the effect of this on the blood is to render it florid; and we may now add, that the chief effect on the air is to abstract a portion of its oxygen, and to substitute about an equal portion of carbonic acid gas. These are the fundamental facts of the *chemistry of respiration*. If we were to enter minutely into the details and explanations of these, we might be led into an interesting field of animal chemistry, which well might occupy half a dozen lectures; but we have so large a quantity of other matter more imperatively requiring attention, that we must dismiss the subject with a few cursory observations.

There are two views which have been taken of the use of the air which passes in and out of the lungs in respiration: one is, that its

oxygen combines with, and carries off merely, a certain quantity of carbon, which the lungs are, by a real secreting power, continually separating from the blood. A superfluity of this carbon is supposed to distinguish venous from arterial blood, it being acquired in the greater circulation through the system, as arterial is converted into venous blood, and thrown off by the pulmonary circulation, when venous blood is again made arterial. This supposes the air to be little more than a broom sweeping, or a current washing, away a carbonaceous excrement thrown out on the bronchial surface; and in confirmation the black matter sometimes expectorated, and that almost always found in the bronchial glands and other corners of the lungs, have been adduced as instances where the air had not swept or washed clean. But there are many, and some I think conclusive, objections against this view. It is opposed by the chemical fact, that simple carbon, where it so excreted, could not combine with oxygen at the temperature of the lungs. It is also opposed by the fact, that the simple contact of air with the blood, without any structure to separate the carbon from the latter, is insufficient to effect the same change in the blood and in the air, as that which takes place in the lungs. Moreover, it is found that not only is no secreting structure necessary, but the change takes place in spite of an intervening membrane, such as a bladder, gold-beaters' skin, or even a thin lamina of Indian rubber. These and many other facts are quite inconsistent with the supposition that carbon is first separated from the blood, and then united with the oxygen of the air.

Of the black pulmonary matter we may find a sufficient source in the blacks and soot from our candles and fires, of the abundant presence of which in the air which we breathe, our linen, our ceilings, and every thing white that is exposed to it afford sufficient evidence. The lungs act as a filter for these impurities contained in the air, which, being insoluble, are not absorbed away, but accumulate in the tissue where they have become entangled.

The other view of the chemistry of respiration is, that the oxygen which is missing in respired air is absorbed into the blood, which throws out at the same time about an equal quantity of carbonic acid gas. The blood is thus arterialized, and, by the oxygen which it contains, rendered fit to excite and nourish the tissues, of which it is the proper pabulum; but in this office, under the control of an unknown vital influence, the oxygen exerts its chemical affinity, and abstracting from the blood a certain quantity of carbon, becomes carbonic acid; and the blood is thus again changed from arterial to venous. The chief characteristic, therefore, of arterial blood is, that it has *oxygen*,—of venous blood, that it has *carbonic acid*, combined with it; and the substitution of one principle for the other will effect the conversion of one kind into the other. This view is supported by a great many remarkable facts, and I know of none which really opposes it.

The change of blood in the lungs is essentially a chemical process, to promote which the mechanism of respiration is eminently

adapted ; but we find the same change to take place out of the body, and to be influenced as chemical processes generally are. The change of the blood in the greater circulation, from arterial to venous, is also partly chemical ; and the affinity which we have adverted to, of oxygen for carbon, is a chemical force that is no doubt active, but this is probably greatly under the control of the vital powers ; and I hold it to be a beautiful proof of the useful subserviency of inorganic to vital chemistry, that this affinity, which we know to be merely chemical, must tend to promote the formation of urea, uric acid, ammonia, gelatine, and other matters containing less carbon than the principles of the blood from which they are formed, which are more properly the products of vital organs, and the necessity of whose generation in the healthy state is obvious from the constancy and regularity with which it is carried on. We have no time to trace further the interesting relations that thus declare themselves in the chemical and vital laboratory of the body ; but I would point out, as one pretty obvious corollary from them, that the function of the lungs stands in a relation peculiarly close with those of the great secreting organs of the body—the kidneys and the liver.

We cannot enter on the subject of the composition of the blood ; but I would call your attention to the remarkable physical difference which we have already noticed between venous and arterial blood—that of colour : that of venous is the most intense dark red—so dark that to reflected light it is almost black ; but by transmitted light, it is of the finest crimson. Arterial blood, on the other hand, is of a bright crimson by reflected light ; but it is of a dingier colour and more opaque than venous blood by transmitted light. This fact led me to the suspicion that the apparent brightness of arterial blood might be owing to the production of a white opacity in it, which would have the effect of reflecting white light through a thin film of the deep crimson. On closely examining the change of colour, whether produced by air or by the addition of saline matter, I have found that a certain white opacity always accompanies the brightening of blood : thus, if you closely watch this coagulum of dark blood when I drop it into this saline solution, you will see that it becomes first white and opaque at the edges ; and this whiteness extending into the mass, shines through the deep crimson colouring matter, and gives it the florid hue. A thin drop of dark blood on a glass, viewed with a lens, appears transparent at first, but gradually as the air begins to act on it, or instantaneously on the addition of a grain of salt, a cloud of opacity is seen in it, which, when seen by reflected light, gives the florid colour of arterial blood. Seen through a good microscope, this opacity is observed to be caused by a myriad of distinct globules, smaller than those of the blood, apparently of the size of the central globule, and of those seen in serum. I have arranged this microscope that you may see this after lecture. The oxygen of the air, or the salt, appears to have the power of giving density or distinctness to the white organic particles contained in the blood, or even to increase

the number of them ; and these particles, acting as so many little mirrors, which reflect light through the colouring matter, give it the brightness so characteristic of arterial blood. Thus the change of colour appears in a simpler light, and one less calculated to mislead, because we are thus reminded of other changes in condition which accompany it. I should not have introduced this matter, but that it serves to show the utility of bringing all our senses to bear on the investigation of a subject—a leading principle in these lectures.

To return to the office of the lungs :—we have no evidence that there is any apparatus organized peculiarly for the absorption of oxygen into the blood, or the exhalation of carbonic acid from it. We have noticed that the displacement of carbonic acid by oxygen takes place in blood out of the body ; that the intervention of a membrane does not impede it ; and it becomes a matter of obvious inference, that the distribution of the blood in millions of capillaries around the air-cells and tubes, with the thinnest possible membranes only separating it from the air, is only an admirable mechanical contrivance for rendering the chemical action between the air and the blood as complete as possible. The fact that air can pervade a membrane was long doubted ; and this doubt was considered, by a great many writers, a sufficient reason for rejecting the view which we are considering ; but this doubt was not well founded ; the facts described by Priestley and John Hunter being enough to remove it. These found that venous blood became florid, although contained in a bladder or goldbeaters' skin ; and Goodwyn rendered the blood in the jugular vein of a living rabbit florid by blowing on it. I had the satisfaction of setting this question still further at rest, by showing, many years ago, that not only was the blood changed in colour, but that the air received carbonic acid from it, and consequently, that the air must have exerted an action on the blood through the membrane. Since I made this experiment, the researches of Dutrochet and Mitchell have developed the fact of the passage of gases or liquids through membranes into a general principle : and one which I believe may be referred to a still simpler law, that of the intermixture of different gases, as developed by Dalton and Graham, and which depends on the comparative elasticities of different kinds of matter. All these steps in science, which we cannot dwell on longer, have dispelled many difficulties which used to beset the theory of respiration : and the further results obtained in the experiments of Edwards, that azote, as well as oxygen, is sometimes *absorbed*, especially during the winter, and sometimes *exhaled*, leave no doubt that gases, as well as liquids and solids, may combine with, and be separated from, the components of the body.

A few other points of adaptation of the minute structure of the lungs to promote the chemical action between the air and the blood, are worthy of notice. The air-canals are lined throughout with a mucous membrane. This, in the trachea and larger bronchi, is not

only of a considerable thickness, but is furnished with numerous distinct follicles, which throw out abundance of the peculiar slimy albuminous secretion called mucus. In the smallest bronchi these follicles cannot be detected, and it is uncertain whether they exist; but the membrane is every where moist and slippery, as if covered by a thinner mucus of the same kind. The utility of this secretion is obvious, in protecting the membranes and tissues of the interior from divers irritations of extraneous matter, from excessive dryness, cold, or heat of the inhaled air; and hence the need of its being thicker and more abundant at the commencement of the canal, where these irritations would exert their fullest effect. But I believe this mucus, in the finer tubes and their extremities, to be useful in another way, in promoting, by its chemical quality, the transfer of oxygen from the air to the blood. Fourcroy long ago showed that the bronchial mucus had, in a signal degree, the property of absorbing oxygen and of yielding it again to other substances, such as metals; hence it is the most effectual medium for killing mercury. I think, therefore, that it is reasonable to suppose the mucus with which the bronchi are bedewed, to aid in facilitating in the oxygenation of the blood. A remarkable phenomena has been lately observed with regard to the mucous membrane of the bronchial tubes of animals recently killed. When it is examined under water, through a microscope, the water is seen to be thrown into distinct and very rapid vortices or currents, which continue with great vivacity for a considerable time, even after the membrane is quite cold. These currents were ascribed by their discoverers, M. Purkinjie and Valentin, to the movements of very fine hairs, or *cilia*; and as they are seen in the gills of some aquatic animals, as the common musclee, in which they obviously serve the purpose of respiration, by keeping up a fresh supply of water to the brachial vessels, they suspected them to exert some similar effect on the air in the bronchial surface of the mammalia. But Dr. Alison has well remarked, that as the same currents are to be seen also in the mucous membranes of the genital organs of female animals, it is more reasonable to suppose, that the object of the movements that produce them is rather the equal diffusion of the mucus, the uniformity of which may be essential to the effectual performance of the functions of these organs. The same distinguished physiologist considers the opinion of Purkinjie and Valentin, that these currents are produced by *cilia* in motion, as too hastily assumed, their existence in the air tubes having never been proved; and he is inclined rather to ascribe them to a vital attraction and repulsion—a principle the existence of which he has brought many ingenious arguments to prove, and one which, if it do obtain, must have a large share in the motions and functions of the body.

LECTURE III.

Vital relations of Respiration—Respiration an instinctive action—Its relations to the Nervous System—Nerves of the Lungs—Nerves of the Muscles of Respiration—Mutual dependencies of the Lungs, the Nervous System, and the Heart; illustrated by a Diagram—Asphyxia, its causes and pathology—Why the Circulation ceases in Asphyxia—Dr. Alison's Experiments—Succession of Death through the Organs—Hibernation—Various effects of Cold on the Functions.

WE have thus briefly surveyed the mechanism and the chemistry of respiration; it now remains for us to consider the function in its vital character—the relations in which it stands to the peculiar attributes of life—and why it is so essential to the life of the body at large. What is the cause which determines the respiratory movements? Is it something inherent in the muscles concerned; or are their actions dependent on a sensation, or other stimulus of the nervous system? Is respiration a voluntary or an involuntary act? How is the act excited first in the newly-born infant? Such are some of the questions that have occupied the wits of great physiologists of many ages. They shall not detain us long. Time will not permit us to run through the diversified answers which successive authors have given to these questions; it will be enough to develop that which, as the most consistent with physiological, and especially pathological facts, will be most useful to us in our subsequent allusions to the function as related to life.

That respiration is not, properly speaking, a voluntary act, is obvious, inasmuch as it usually takes place without any act of the mind, even in coma and complete stupor, when the mind is incapable of willing. But we know, also, that we can at pleasure control respiration; we can increase it, quicken it, and for a certain time stop it. All the muscles of respiration are, therefore, subject to the will, although acts of volition are not necessary to their *regular* action. What is it, then, that excites them in this regular action? We may find out, by trying to its utmost the power of our will to stop respiration. We stop our breath for a little while by a direct control of the muscles of respiration; but soon we feel a sensation of oppression, which irresistibly throws these muscles into action; and the only way in which we can still stop our breathing is by closing the glottis, and keeping the diaphragm pressed upwards by the contraction of the abdominal muscles. In the course of a few seconds, however, the sensation of uneasiness in the chest becomes intense, the respiratory muscles make violent efforts, and the voluntary act which stops their play is soon conquered by the sensation becoming insupportable. It is stated by some writer on legal medicine, that a man once intentionally put an end to his existence by holding his breath; but it is not likely that the death was one of simple asphyxia, because the mental act of holding the breath could not continue when that mental stupor, that *defectio animi*, came on, which always precedes this kind of death. But,

as we shall afterwards see, the act of holding the breath causes such congestion and obstruction in the circulation, as might readily lead to a fatal result by other ways, such as hemorrhage in the lungs, syncope of the heart, or apoplexy, in those individuals who are predisposed to these affections.

I would, with Dr. Alison, class the action of ordinary respiration among those which are called instinctive, as being excited by an impression of which, in its ordinary degree, the mind is not conscious. Of exactly the same kind is the action of the eyelids in winking; but similar instinctive motions, equally unattended by any distinct volition, are excited in other voluntary muscles, by particular impressions or sensations—as vomiting by a certain degree of nausea, or by tickling the fauces; laughter by tickling under the armpits; the rapid withdrawing the hand or other part from fire burning, or any thing else severely painning it; and the very action of taking a breath on the sensation of sudden cold applied to the face or surface. The great distinction between these instinctive acts and those which are voluntary, is, that instinctive acts, however complex, are uniform in all individuals, and perfect from the earliest age; whereas the simplest voluntary acts which are preceded by a mental process, vary in different individuals, are imperfect at first, and require practice and education to make them perfect. Thus the child breathes well at first, but does not walk or talk well till after long practice. The sensation which prompts the respiratory act is one which remains after common sensibility ceases, and when the mind gives no sign of activity, as in coma, or in profound sleep; and it has been proved by the direct experiments of especially Legallois and Flourens, that of all parts of the nervous centres, the medulla oblongata is alone essential for its continuance. The brain and cerebellum may be removed without stopping it; but whenever the medulla oblongata is removed, or materially injured, respiration immediately ceases. So respiration is stopped by those influences which destroy sensibility; but it requires a greater degree of them than that necessary to destroy common sensibility, the sensibility which prompts the respiratory act being one of the last to abandon the body. Thus opium in a large dose first throws a person into that state of stupor in which he is insensible to common pain, pinching, &c., but he breathes still. He breathes, it is true, in a laborious and imperfect manner; and although the impression which is acting on the remaining sensibility must be of a very intense kind, it becomes powerless as this is lost; and the respiration then ceases. A similar result is produced by apoplexy, or any injury which presses on or hurts the medulla oblongata. In apoplexy the pressure is more or less general on parts of the brain connected with common sensations, and these go first; whilst, if the pressure is considerable, the sensibility connected with respiration follows, as in the case from opium. But in direct injuries to the medulla oblongata, or upper portion of the spinal marrow, as where the neck is broken, respiration is destroyed from the first, and death is im-

mediate. But the effect of these injuries or influences may be two-fold : it may paralyse those nerves which give sensibility to the lungs ; and it may paralyse those motory nerves which convey to the muscles of respiration the stimulus of the sensation or impression ; and this brings us more closely to the relations which subsist between the lungs and the nervous system.

The nerves of the lungs (to which I did not advert in speaking of the mechanism) are from the eighth pair, or par vagum. These nerves, just before they enter the lungs, inosculate with the great intercostal ; but, according to Reisseissen, the par vagum is the only nervous *trunk* distributed on the lungs.

Now the eighth pair appear to be nerves of motion as well as of sensation. Their sensitive function we have already dwelt on ; and we find in the bronchial muscles, and especially in those muscles which open the glottis at the moment of inspiration, and are supplied by the recurrent branch of the eighth pair, the objects for the motory office of these nerves. The share which this nerve has in transmitting to the sensorium the impressions which excite the muscles of inspiration, was manifested in some recent experiments by Dr. M. Hall and Mr. Broughton, in which pinching the nerve was immediately followed by an effort at inspiration ; and that this does not depend on the motory functions of the nerve, is obvious from the fact, that the same effort is excited when, after the division of the nerve in the neck, its upper segment only is compressed, which is exactly what occurs with nerves of sensation, while the contrary happens with motory nerves. The division of this nerve does not at once destroy the sensibility on which the respiratory movements depend, but it greatly impairs it ; for mechanical irritation of the bronchi will not then excite coughing, and expectoration consequently ceases. Another effect of the division of the nerve has been noticed by Mr. Swan, namely, that the air which is introduced into the lungs in inspiration, is not duly expelled by expiration, and, accumulating in the cells, causes a certain degree of permanent distension. This implies a degree of paralysis of the bronchial muscles, the contraction of which we have before noticed as a part of the forces which complete the act of expiration, not as being of much force in itself, but as equalizing and aiding the diminution of the volume of the lungs, which is mainly effected by the pressure on them of the diaphragm, and of the parietes of the chest. I would beg your attention to a circumstance which seems to show that this incomplete expulsion of air is also to be greatly ascribed to a loss of sensibility in the bronchial membrane.

If, after an ordinary expiration, we hold our breath until the sensation in the chest becomes painful, and then breathe, the first act is usually a quick, but short expiration, followed by a deep inspiration, as if the lungs were even more anxious to *expel* the remaining foul air, little as it is, than to draw in pure air. When we hold our breath after inspiration, the disposition to expire air is of course much stronger ; and the act of merely breathing out a little slightly

relieves the sense of oppression, and enables us to hold on a few seconds longer without taking in more air. These things seem to imply that the bronchial membrane is irritated by the presence of foul air, and seeks to get rid of its presence, both by the contraction of its own muscles, and by exciting those distant muscles concerned in expiration, which instinctively obey the peculiar sensation or impression which is present. It is not unimportant to notice these circumstances as showing that expiration is not merely mechanically necessary to prepare for inspiration, but that it is also a vital action under the influence of its own sensibilities and corresponding forces, as we have seen with respect to inspiration.

We have seen that the division of the eighth pair in the neck, although it greatly impairs respiration, does not arrest it; and a sufficient explanation of this may be found in the inosculaton of these nerves with the intercostals, through which the impression which excites the respiratory act may still, in a certain degree, be transmitted to the medulla oblongata.

The relations between the lungs and nervous system that we have hitherto considered, are chiefly those of sensation; but there is an indirect relation between the lungs and certain motory nerves, which is equally important in maintaining the circle of communication between the lungs and the mechanism by which they are filled and emptied. The chief motory nerves supplying the muscles of respiration are the phrenic, which go to the diaphragm, the intercostal twigs of the spinal marrow to the intercostal muscles, the spinal accessory nerve to the sterno-mastoid and trapezius, the external respiratory to the serratus magnus, various of the cervical nerves to the scaleni, and other muscles more or less concerned in raising the thorax. It has been found by experiment that the section of these several nerves, or of the spinal marrow above their origins, suspends the action of their respective muscles in the respiratory movements. There are, besides some motory nerves, which co-operate, not through muscles which alter the capacity of the chest, but through those which keep open the canals through which the air passes into the chest. Of these the recurrent branch of the eighth pair supplying the crico and thyro arytenoid muscles which keep open the glottis, and the portio dura of the fifth pair through which the nostrils are dilated, are the chief; and of these it has also been found by experiment, that a section of the nerves prevents the free opening of the respective passages. Dr. H. Ley has pointed out pathological facts which illustrate the same point with regard to the recurrent nerves, in the case of spasm of the glottis from tumours under the upper part of the sternum compressing these nerves, which generally antagonise the superior laryngeal nerves which influence the muscles closing the glottis.

The various motory nerves concerned in respiration, may be excited by other sensations besides that from black blood and foul air in the lungs: thus cold water dashed on the face will cause a deep inspiration; tickling the nostrils will cause sneezing; irritating the glottis, coughing; pain, sobbing, &c. We avail ourselves of

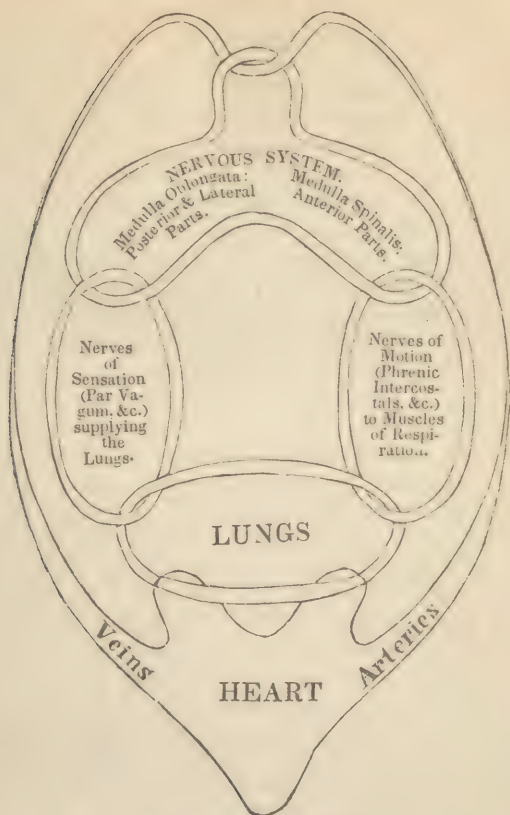
these sympathetic associations of nerves and muscles to excite respiration in new-born infants, and to restore it in cases of suspended respiration. That there is sufficient ground for constituting these various motory nerves into a system distinct from those which move the muscles in obedience to the will, as supposed by Sir C. Bell, is very doubtful; and the reason assigned by him for their exciting and combining the respiratory movements, that they originate exclusively from the lateral columns of the spinal cord, from which also the great sentient nerve of the lung, the eighth pair, arises, is, as Dr. Alison remarks, by no means established. It would be easy to prove, by a reference to the anatomy and functions of the nerves, that two nerves arising from one root do not necessarily act in concert. We must, therefore, add that the reason of the concert is not explained; but its cause, its object, is obvious, as Sir C. Bell himself observes, in the nerves and muscles employed to aerate the blood, "being put under the guidance of a sensibility more certain and more powerful in its effects than the will."

There is thus made out this double relation between the lungs and the nervous system—that of *sensation*, acting from the lungs to the medulla oblongata; that of *motion*, from the nervous centre, through various nerves, to the muscles of the chest which move the lungs. The diagram given further on exhibits these relations. That there are, besides these, other *occasional* relations between various parts of the nervous system and the lungs, capable of *influencing* the functions of the vessels of the latter, is very probable; but that these relations are constant, or in any way necessary to the function of respiration, or to the nutrition of its organs, as supposed by Dr. Wilson Philip and others, is a matter of mere hypothesis, unsupported by any unequivocal fact, and rather opposed than otherwise by the known attributes of the nervous system. It might be interesting, but I doubt that it would be profitable, for the purposes of these lectures to enter further on these matters.

We have thus seen how the function of respiration hangs, as it were, on that of the nervous system: we have now to trace the converse link—how the nervous function is also dependent on respiration. This link, most important in itself, and equally important in spreading the effects of respiration through the system, is constituted by the heart and the double circulation connected with it. All that we learn from the mere mechanism of the heart and its sets of tubes is, that it is admirably adapted to carry the blood in one direction—from the right auricle to the right ventricle, from the right ventricle to the pulmonary artery, which subdivides again and again until its branches are of that size which constitutes the capillary blood-vessels of the lungs. These form a network around the air-cells and minute tubes, from which network the many smallest branches of the pulmonary veins arise, which unite into fewer larger ones; these, again, converge into the four pulmonary veins which bring back the blood to the heart, now entering the left auri-

cle ; from this it passes to the left ventricle, and thence is distributed by the aorta and its various branches throughout the system. These branches subdivide, until, as in the lungs, they form the capillary network from which again arise the veins, which converge into trunks ; all these unite in the venæ cavæ, which, entering the right auricle, completes the circulation. All this course is plain from the mechanism of the valves of the heart and veins, and can be imitated in the dead body, by injecting liquids through the vessels. This is the *hydraulic* apparatus of the circulation, and, as such, it is regulated by hydraulic laws, which deserve their share of our attention, and will be further considered in the third part of this course, when we come to treat more particularly of the heart and great vessels.

But we have now to think of the vital properties that are added to the mechanism, the contractility of muscular fibre, and the relations in which that contractility stands to the quantity and quality of the blood that excites it, and the relations which subsist between it and the nervous system, which can influence it. We have also to think of the blood, not merely as an inert liquid, passively propelled through pipes, but as a liquid teeming with chemical and vital properties, which are continually acting on the vital properties, and, through them, on the mechanical powers, of the various solids through which it passes. Between all these properties there are certain relations or proportions which may be called healthy or natural, inasmuch as they tend to support each other with such an even balance as leads to durability and permanency. Thus, for the proper or natural action of the heart, its right cavities should receive a due supply of venous blood, which being their proper stimulus, excites them to propel it into the pulmonary artery ; its presence there excites, in the nervous system, the impression which leads to the movements of respiration ; these, by bringing the air to act on the blood, renders it arterial, and thereby fitted to return by the pulmonary veins to the left cavities of the heart, of which, thus arterialized, it is also the proper stimulus ; from these it is propelled through the various arteries to all parts of the body, of whose functions and textures it is the proper support and pabulum. Among these parts which thus require the supply of arterial blood, are the substance of the heart itself, which thus supports its own function, whilst its function is necessary to effect this support : the nervous system, which as it is necessary to maintain the function of respiration, so in receiving arterial blood it becomes through the heart dependent on that function ; and the muscles of respiration in like manner depend on their own work through the heart, which supplies them with its product. Thus is the chain of mutual connexion and dependence complete, and it may be seen in this diagram how any break in this chain must destroy the order of the whole arrangement, and arrest that circulation of influences which is the characteristic of life. The dependence of the heart on the nervous system through the lungs, is sufficiently plain in this diagram ; but you may see that



THE CHAIN OF LIFE.

there is an attempt also to represent that the action of the heart is otherwise independent of the nervous system, although it may be influenced by it. Thus the nervous system hangs on the heart for its supply of blood; but the heart hangs on the nervous system only through its link with the lungs. Substitute for this link artificial respiration, and you may then *quietly* cut away the nervous system without disturbing the heart's action. But if you do *violence* to the nervous system, you may shake and disturb the heart also. You may find this diagram useful in studying the causes of asphyxia, syncope, and other kinds of death.

Having thus analyzed the relations, vital and physical, of the chain or tripod of life, the heart, the lungs, and the nervous system, the nature of *asphyxia*, which consists in an interruption to those relations, will be generally intelligible. The etymology of asphyxia implies the very converse of the pathological condition to which

the name is applied. It means no *pulse* ; whereas the condition to which it is applied, consists essentially in no *breath*. It is true, the pulse must soon cease, as you perceive, when there is no breath ; but the characteristic of asphyxia is, that there is pulse when breathing has ceased. The name is, therefore, obviously improper. With this understanding, however, and using the term under the figure of *lucus à non lucendo*, we inquire what are the causes of asphyxia, that is, what will stop the breath ? We see in the diagram an answer with regard to the nerves and their centres,—any thing that cuts off communication with, or any thing which destroys the function of, this system, whether by physical injury to their mechanism, or by poisons acting on their vitality. But, besides, we have the mechanism of respiration,—any thing which stops that, any thing that renders its movements ineffectual, by cutting off the supply of pure air, annihilates the function of respiration, and stops its essential important effect, the arterialization of the blood. Well, now, let us trace the successive phenomena of this stoppage of respiration, how they follow one another. The oxygenation of the blood in the lungs being arrested, this blood necessarily remains venous ; and from the mechanism we should expect it to pass on in this venous state to the left cavities of the heart : a little does so pass ; but it is only a little ; the greater portion is arrested at the lungs, waiting, as it were, for the supply of air, and being refused any onward progress, the accumulation of blood distends the right cavities of the heart, then the venous trunks, and a great portion of the venous and capillary system ; the left cavities and arteries being nearly empty. It was supposed, by Goodwyn and Bichat, that the black blood became distributed through the arteries, and acted on the parts through which it passed as a sort of poison, that suspended their functions, and that thus the death which began with the lungs was transferred throughout the brain and other parts of the frame, destroying speedily the heart also. But Dr. David Williams, of Liverpool, first showed that very little black blood passes to the left side of the heart, but it is somehow arrested in the now motionless lungs. And Dr. Kay found that slowly injecting large quantities of venous blood into the carotid artery of an animal, did not suspend the function of the brain, nor cause the loss of sensibility which attends asphyxia. Dr. Edwards further proved that the irritability of the muscles of cold-blooded animals is preserved longer when only venous blood flows through them, than when they receive no blood at all, which could not be the case if venous blood had a positively noxious quality. Hence it is probable that the sudden failure of animal life in asphyxia is caused by the great want of supply of blood in quantity, rather than by its venous quality, and that this obstruction in the lungs destroys by soon arresting the circulation throughout the body. Hence the latter phenomena of asphyxia, the failure of the senses, sometimes with slight convulsions, resemble those of syncope, in which the heart at first ceasing to act, the circulation is at once stopped, and the functions of the nervous

system also fail. Although, however, the defective quantity of the blood may be the chief cause of the failure of the functions in asphyxia, I cannot but consider the venous quality of that which does circulate, or stagnate, as a concurring injurious influence. This blood, deficient in oxygen, and abounding in carbonic acid, produces in the brain the same symptoms that Nysten obtained by injecting carbonic acid into the carotid artery; and whether this venous blood is supplied directly through the arteries, or made to stagnate in the vessels of the head, by an impediment to its progress onwards through the veins to the lungs, all forms of asphyxia seem to agree in this condition—the presence of black blood, and of only black blood, in the brain and other organs.

A question of great interest arises from the view of the pathology of asphyxia, which the experiments just alluded to develop. The venous blood, not finding oxygen in the lungs, will not pass. What is it that stops it? The right side of the heart continues to beat, even after the left side, no longer receiving its accustomed stimulus, has stopped. The obstruction must, then, be in the lungs; and Haller supposed that the absence of the alternate motions of inspiration and expiration, which must assist the passage of the blood onwards through the capillaries, is sufficient to account for the obstruction. But Professor Alison has lately made experiments which throw doubt on this explanation. He found that when an animal is confined in azote until its breathing becomes laborious, and then killed instantaneously by concussion, the right side of the heart and the veins were as much distended with blood as where the respiratory movements had ceased. Here, he maintains, were the mechanical motions of the lungs, described by Haller, kept up to the last, but the pulmonary obstruction nevertheless existed. Dr. Alison views this obstruction as one of many other phenomena, which seem to prove that the motion of the blood in the capillaries is independent of any contractions or movements in the vessels themselves, or in any of the living solids: that it is caused in great measure by certain vital attractions and repulsions subsisting between the particles of blood and their containing vessels; and that these vital attractions and repulsions are greatly modified by the chemical changes which the blood may undergo in the various organs. In the lungs, the absorption of oxygen promotes through these powers the passage of blood through the capillaries. When oxygen is not present, this power of capillary circulation ceases, and the blood necessarily stagnates. A single breath is, however, enough to set it in motion; and by artificial respiration the circulation of the blood may be restored after the heart has ceased to act, and when it (the heart) is re-excited by the oxygenated blood reaching it, propagated by these other powers. The correctness of this explanation is involved in the question as to the existence of these powers of vital attraction and repulsion, which is far too extensive for us to enter into here. The view that there are *physical* properties *peculiar to living structure, vital attraction and repulsion*, as we

know there are *peculiar chemical properties, vital affinities*, is quite philosophical in principle ; and the number of facts which Haller, and many of his countrymen since his day, have adduced in support of this view, are highly deserving of your attention.

But I confess that I wait for further arguments before I can be quite convinced that this view is absolutely necessary to explain the phenomena of asphyxia. 1st. I do not think that Dr. A.'s experiments are altogether conclusive against the explanation of Haller—that the obstruction of the blood in the lungs is caused by a stoppage of the *natural* motions of respiration. Dr. A. substituted azote for air, and supposed that the mechanical motions of respiration of this would be the same as of air ; but from what I have before said, it is probable that air, deficient in oxygen, cannot be so freely respired as common air. Air that is kept until much deteriorated in the lungs, causes a forced expiration ; and the reception of the same air again must very promptly excite the same action, until oxygen is supplied ; so that the respiration of azote must consist in a preponderance of expiratory over inspiratory efforts, which would tend to retard the passage of blood through the pulmonary vessels, instead of promoting it, as the regular succession of equal in and out breathing does. I do not, however, suppose that this is more than one of the causes which obstruct the progress of blood through the lungs. But, 2dly, there is a contractile power in the blood-vessels which is capable of obstructing, although it cannot promote, the motion of the blood in them. The capillaries themselves do not seem to possess any power of this kind, but the larger vessels (both arteries and veins) have it, and are seen to show it on the application of an unusual stimulus. Now there seems to me nothing unreasonable in the supposition that venous blood is such a stimulus to the small branches of the pulmonary veins, which, being adapted to carry only arterial blood, may thus refuse a free passage to unaerated blood. These considerations will, I believe, go some way in explaining the impeded pulmonary circulation in asphyxia. The passage of blood is not wholly arrested, but it is impeded enough to cause great distension of the right ventricle ; and this distension, as we shall hereafter find, impairs the power of its propulsive action, by keeping open the tricuspid valves enough to cause regurgitation and venous pulse. This venous pulse doubtless answers the salutary purpose of giving a certain motion to the blood stagnating in the veins, which might otherwise coagulate ; but it must add to the difficulties against the onward progress of the blood. The left ventricle receiving so little blood, and that not adapted to excite it to its usual activity, and sending so little of the same fluid through the coronary arteries to revive its substance, soon ceases to act ; the arterial circulation also ceasing, those parts die which depend on its support, the nervous system dies, the muscles die, and at length the right side of the heart, failing to receive its pabulum through the coronary arteries, dies ; and last of all, no longer receiving the vivifying operation of the functions and of motion, the blood coagulates, and dies also.

There is a condition illustrative of the relations of circulation and respiration, which is worthy of attention, as it presents us with a sort of separation of these functions—*hibernation*. When hibernating animals are exposed to a certain degree of cold, instead of exhibiting that power of reaction, of increased activity of respiration and secretion, which enables other animals to generate more heat, and thus to preserve for a time their temperature, they become torpid, gradually lose their temperature, the respiration becomes more and more rare, and at length ceases; and the circulation becomes very slow and very languid. In these animals, cold suspends entirely the animal functions, and greatly reduces the organic, but does not destroy the irritability of the heart, which, continuing to beat, circulates slowly a little venous blood; and this little, which would be wholly insufficient to support the functions when warmth develops them into activity, sustains them in the degree of vitality to which cold has reduced them. Warmth reapplied is the most effectual key to the locked-up properties of life: as the animal becomes warm, its sensibility returns; it feels the want of breath, muscles obey the call, respiration begins, circulation is quickened, secretions are excited, the self-warming power is regained, and the animal is raised, in the course of half an hour, from a state of animation below that of a cold reptile, a condition more like vegetation, to the vivacity and activity of a warm-blooded mammal.

When a hibernating animal is in its most perfect state of torpor, there is apparently no respiration, and the blood which circulates is only venous. Now as by our diagram we have represented respiration to be the chief link by which circulation depends on the nervous system, this link here no longer existing, we should expect that the dependence which it maintains will be in a measure suspended. Accordingly, Dr. M. Hall found that the circulation of an animal in this state went on for nine hours after the gradual but complete destruction of the brain and spinal cord. Observe, however, that even in this state the nervous system could still *influence* the circulation; for a violent injury, such as crushing the brain, would arrest the circulation by a positively noxious influence, just as would happen from crushing a limb. So also sensibility is not entirely destroyed, but becomes of a latent and conservative kind, and is capable of being excited and developed into greater activity by strong impressions, or those of a destructive tendency. Thus even an intense cold, such as that of zero, will be felt by a hibernating animal; the sensation will excite the dormant functions, and the animal will awake from the intensity of the same agent that brought him to this state of torpor. The effect of gradual cold seems to be to lower the function of the nervous system in such a way that the lungs and other parts do not feel the want of arterial blood, but in their degraded state to be content with merely venous blood. The effect of sudden intense cold is different, but it illustrates the same point. A warm-blooded animal, suddenly exposed to intense cold, as by being immersed in ice-cold water, is brought

into such a condition that the arterial character of the blood is not needed, and the veins return it to the heart in its florid state ; those functions which need arterial blood are paralyzed, and the chemical change from arterial to venous, itself also partly dependent on temperature, is therefore suspended. The paralyzing effect here, however, extends to the heart, which, although receiving arterial blood, cannot obey its stimulus.

The diminution of animal functions which hibernating animals present in the highest degree, is to a certain extent exhibited in most other animals. Thus on a severe winter's day, we see crows and other birds perched still and almost inanimate on the trees ; the hunger which they must feel not impelling them to exertion. Even domestic poultry prefer to roost half their day, without food, to the exertion of wandering as they are wont to do in search of it. Nay, the longer sleep which the protracted absence of light in the winter brings on animals, is wisely intended for the same purpose as hibernation—to lower those functions the activity of which would cause acute suffering at this inclement season, and for the maintenance of which there is no longer an external supply. Man, and the animals which he domesticates with him in his artificial state, exhibit the least of this natural adaptation to seasons : for this he substitutes artificial protections and seasons ; and thus keeping the functions in their condition of full activity, he incurs, on any accidental neglect of his artificial means, risks from sudden transitions or exposure, from which other animals are comparatively free.



LECTURE IV.

Physical Examination of the Chest—Modes of Studying the Topography of Organs—
 Examination of the Chest by inspection and feeling—Natural form of the Chest—
 Postures for inspection—Examination of the motions of the Chest by sight and touch—
 Varieties of Respiration—Mensuration of the Chest—Measurement of the Air respired
 —Limitation of the Signs obtained by sight and touch—Need of another Sense—
 Modes of studying the Acoustic Phenomena of the Chest.

HAVING now anatomically and physiologically gone through the general structure, functions, and relations of the chest and its organs, we have next to study these subjects as physicians ; and you will immediately see how essential our previous knowledge is to prepare us for this study. We have to find out the signs or symptoms through which, in the living body, we can judge of the condition of the various parts of the structure, and of the performance of their several functions, and thus through which we can distinguish health and disease. This department we will, if you please, call EXAMINATION OF THE CHEST ; for although it is the organs within it, rather than the chest itself, which we generally wish to study, we must

never forget that the chest is, as it were, an outer coat of these, organs, fitted to them, and partaking of their shape, motions, and sensibilities ; and as in health these all work and feel together, so in disease we must never lose sight of their mutual dependencies and connexions. Now as we have considered the properties of the chest and its organs under the two heads, physical and vital, so you see that in my prospectus I divide *Examination of the Chest* into two kinds :—1. As practised PHYSICALLY by vision, tact, and hearing. —2. THROUGH FUNCTIONS, or through those more complex properties or actions which depend on vitality superadded to physical structure. These two kinds, although studied separately, must be used jointly.

We examine the chest PHYSICALLY, through those properties of form, size, proportions, and relative position of its parts, at rest and in motion, which are appreciable by our external senses. Now, gentlemen, mark me ; you cannot get on well in the physical examination of the chest without a *topographic* knowledge of the organs within it : you must know where each severally lies and reaches with regard to the exterior, so that when you inspect, feel, and listen to the chest, you may be able to map out the general outlines of the organs within.

You will ask me, how are we to get this knowledge ? There are two ways to it ; and I would advise you to use them both. The first and most obvious one is, by attentively observing, every time you see a body opened, the position which the organs hold with regard to the exterior. The minutes which we are apt to consider as time lost, while we are impatiently waiting to see the morbid anatomy within, may be profitably employed in taking a lesson of the anatomy of position. As the dissector's knife cuts the integuments, our eyes may rapidly survey certain external marks which we may be ready to compare with the interior when it shall be opened. There are the nipples, the edges of the pectoral and serrated muscles, the intercostal spaces, which can generally be counted with ease near the sternum, the divisions of this bone, the clavicles and scapulæ, and divers minor elevations and depressions on the exterior, all of which may serve as landmarks to indicate the position or boundaries of the organs and their several parts. The moment the sternum is raised, and before the lungs collapse (which may be prevented by closing the nostrils,) we can glance at the extent to which these organs cover the heart and reach downwards, the position of the air and blood-vessels, the height of the diaphragm, and the abdominal viscera beneath, and we can transfer these various sites to some of the marks or lines of the exterior.

I do not mean to assert that this habit of comparison of the outside with the inside in the dead body will give you a very accurate knowledge of what exists in the bodies of the living ; for besides that there is some variety in different individuals, there can be no doubt that on the motions and properties of life being destroyed some changes may consequently take place in the size and relative

position of the organs, and these may vary according to the mode of death. Thus it is probable that the diaphragm, relaxed by death, permits the abdominal viscera to encroach on the cavity of the chest further than during life ; and the volume and position of the heart and lungs will be affected not only by this circumstance, but by the condition of the circulation and respiration at the time of death, and by the influence of time and temperature on the stiffening of the muscles, and other changes which immediately succeed death. These are subjects which deserve further investigation. In spite of these exceptions, which it is well to be aware of, I repeat that you may gain much valuable knowledge of the topography of organs in the way that I have described, and this knowledge will be of such a personal, practical kind, that it will gradually confer on you a self-correcting tact and insight which you can never gain from mere rules and descriptions.

There is another way to the knowledge of the topography of organs ; it is more exact, but more difficult. It is the *habit* of physical examination ; and thus our study enlightens itself. We cannot dwell on this now, because it comprehends the details which will occupy us for several succeeding lectures ; but I will tell you, by way of encouragement, that after you have carefully examined half a dozen subjects, by the different physical means which we have now to explain, you will have gained results which may supply you with a good general idea of the topography of the living chest. In short, it comes to this—practice alone can make you perfect in this, as in every other study in which the senses are to be exercised.

I have attempted in these diagrams to set before you views of the topography of the pectoral organs, and by giving a transparency both to the chest itself, and also to the viscera, I have endeavoured to represent their form, size, and relative position. Here, for instance, is an external front view of the trunk of a man : it is sketched, as you see, in Indian ink, with all the points, prominences, and depressions, which are presented in the natural contour of the body. But through this exterior you see the lungs, the heart, the liver, the stomach, the spleen, &c. tinted in different colours, and by a convenient transparency in these viscera, you get a general view of the interior—how the several large vessels pass, and how one organ is superposed over another. Thus here are the lungs with the large bronchi coloured light pink, and the liver purple ; and where at the margin of the diaphragm the latter of these viscera comes in contact with the external parietes, its colour is deep and distinct ; but where it rises above into the vaulted hollow of the diaphragm, it is seen in a fainter colour, because it is gradually shaded off by the superjacent light coloured lung. So also the heart, in the small space in which it comes in contact with the chest to the left of the lower end of the sternum, has its crimson colour ; but it also communicates its tint more faintly through the lungs which lie over its whole body, and the vessels which proceed from it, and so on with other organs. Here is another diagram representing the back view of the chest and abdomen. [These diagrams cannot be given in print.]

Now you are not to suppose that these drawings give a very exact view of pectoral topography, for, as I have said before, the parts and their outlines are to a certain extent variable and shifting ; but they will assist you in the two other modes of study which I have pointed out, and they will be of material service to us in our explanations of various diagnostic phenomena of health and disease. You may, too, if you please, take from them the idea of transparency ; and when you examine a chest, try, in a study of external marks, to see the outlines and positions of the organs within.

Well, now let us touch lightly on the examination of the chest by sight and feeling. The patient standing or sitting even, with his arms and legs in corresponding positions, and his chest if possible entirely exposed in a good light, we view it in front, behind, and from above, and mark its form and proportions, and corresponding prominences and depressions. A healthy chest is very nearly symmetrical, the two sides corresponding in shape and size. The right side is, however, almost always slightly larger than the left, especially at its lower portion, where the difference of measured circumference amounts generally to half an inch. This preponderance in favour of the right side is in part to be ascribed to its contents, but it is chiefly connected with a law which pervades the animal creation, which gives a superiority of development to the right side. In some of the Mollusc tribes you see this law prevail to such an extent that the right side is rolled round the left, as in the house snail. There is, on the other hand, in most healthy chests, an advantage on the left side in point of height. The apex of the left lung, and the corresponding portion of the chest, rises a trifle higher than that of the right. I cannot tell whether this is an original condition, or whether it results from the habitual inflated pressure of the stomach and the unyielding mass of the heart on the left side, giving the chest a greater tendency to upward expansion, but the fact is pretty constant. Still these slight exceptions scarcely detract from the general symmetry of the chest, and when known can be allowed for. Where you find any considerable departure from this degree of symmetry, or correspondence between the two sides, you may be pretty sure that there either is, or has been, disease.

I have said that you may inspect the chest from *above*, as well as from before and behind. This may be done when the patient is sitting on a low seat, with his head a little bowed forward, by standing behind him and looking down on the shoulders. You thus get a view of the depth of the chest from front to back, and you may often detect a want of correspondence between the two sides, that is not perceptible by the ordinary modes of inspection.

If the patient's strength do not permit him to stand or sit up, you may inspect the chest when he is lying on his back ; and this may be done not only standing by the side, but also at the foot and at the head of the bed, from which the corresponding motions of the two sides can be better seen.

Now the inspection of the chest is to be applied not only to its

statical condition, but also to its motions; and here it is proper to employ feeling with it. Whilst, therefore, we are inspecting the chest, we desire the patient to breathe in various degrees; and with our hands and eyes on corresponding points of the two sides, we watch and feel the amount and equality of the motions. If the chest is a healthy one, we see the motions as uniform as the chest is symmetrical. The clavicle, scapula, and upper ribs rise; the lower ribs rise and spread; and the abdomen swells as the diaphragm descends at each inspiration. Attentively watching and feeling the chest will also often enable us to trace the limits of some of these movements, so as to indicate the boundaries of the chest. Thus we have before noticed that the lower ribs are pressed outwards, by the displacement of the abdominal viscera at each descent of the diaphragm, and constitute a fulness below the limits to which the lungs descend, with a slight flatness or hollow above. My friend, Dr. Edward Harrison, has studied these marks so successfully, that he can discover in many cases, by simple inspection, the height of the diaphragm and liver. The intercostal spaces are also fit marks for this mode of comparison between the two sides; and there are other useful details, which I cannot now enter into: they are soon learnt in practice, when the principle of the examination is properly understood. Applying the hand in the region of the heart, we feel the relation of the respiratory motions to that organ. After a full expiration the heart is felt beating close under the hand, about the cartilages of the fourth and fifth ribs, as well as under the sternum; but as the ribs rise, and the lung expands in inspiration, we gradually lose the beating, and if it is felt at all, it is now as low as the sixth rib. I shall not dwell longer on the healthy appearance and motions of the chest, but you should take every opportunity to study them; and you will then soon be able to scan them, just as a horse-dealer judges by a few glances of the points of a horse.

We shall notice a few general varieties of respiration, which are to be determined by watching and feeling the motions of the chest. Healthy or perfect respiration is both diaphragmatic and costal; but under the influence of disease the motions may be confined either to the ribs only, or the diaphragm only. Thus, when the diaphragm is prevented from descending by acute pain in it or below it, or by pressure from below, the respiration is wholly performed by the raising of the ribs, and is called *heaving*, *thoracic*, or *costal* breathing. When the ribs, again, are immoveable, in consequence of pain, ossification of cartilages and ligaments, or paralysis of the intercostal muscles, the breathing is wholly *diaphragmatic* or *abdominal*.

The movements of respiration may be *partial*, when one side of the chest is seen to move less than the other, or when a part of one side moves imperfectly; and this partial respiration may proceed from immobility of the parietes, or, as more usually, from impermeability of the corresponding portions of lung, in consequence of various diseases. Thus, when lymph or tubercular matter in the tissue of the lung, and obstruction of the bronchi, an effusion into,

or a contracted adhesion of, the pleura, prevent the inflation and collapse of a part of the lung, the corresponding walls of the chest will be resisted in their motions, and will become fixed in proportion. Thus in phthisical patients we often see the ribs below the clavicles scarcely moving in respiration, and often sunk on one side; in pneumonia and pleurisy the lower ribs are more commonly fixed. Mark further how they are fixed, whether in a state of dilatation, or in one of collapse—whether the affected part remains full after expiration, or whether it is still sunk after inspiration, or whether it is fixed in an intermediate state: you may thus, in certain instances, go some way to distinguish between these different causes of pulmonary obstruction. But more of this by and by.

Examination by tact and vision requires, on the part of the observer, an exact eye, a good perception of shape and symmetry, or, if you will have it phrenologically, full organs of *size*, *form*, and *order*. It is often highly useful in the way of a general survey, preparatory to other more accurate modes of examination.

The plan of measuring the chest is a more exact method of detecting inequalities of size between the two sides of the chest. It is generally practised in this way: a piece of tape is fixed with one end on the spinous process of a dorsal vertebra, and carried horizontally to the middle of the sternum, first around one side, then around the other, in this way. [The method was shown on a plaster figure.] Of course great care must be taken to pass the tape around corresponding parts; and attention must also be paid that the degrees of the respiratory act be the same during the measurement. The most accurate mode is to compare the measurements of the two sides on a full inspiration and a full expiration, as well as in the intermediate state. The process is rather a troublesome one, and we cannot, among private patients at least, use it often; but it sometimes gives results which are well worth this trouble.

Besides external measurement, which is essentially comparative between the two sides, I may notice here a sort of attempt at internal measurement, by noting the quantity of air that can be *exhaled* or *inhaled*. Mr. Abernethy proposed to judge of the capacity, and consequently the soundness, of a man's lungs, by measuring how much air he could throw at a breath, after a full inspiration, into a jar inverted over water. Here is an apparatus which belongs to Dr. Green, of Great Marlborough Street: it measures the quantity of air taken in at a full inspiration. You see, when I close my nostrils and inspire through the tube, the water in the glass jar rises in proportion to the quantity of air which I draw in, and this is measured by the cubic inch scale on the side of the jar. There are other contrivances of a similar kind. The chief objection to them is that their indications are affected not only by the capacity of the lungs, but also by the strength of the respiratory efforts. They are *dynamometers* for the muscles of

respiration, as well as *pulmometers*; and a weak, delicate, or nervous person, with sound lungs, tested by them, would be placed below a phthisical or pleuritic patient whose muscular energies are still considerable.

So much for *seeing* and *feeling*. They may often do something, but you will readily perceive that they will seldom inform us of the nature of the obstructions which they can discover, and they cannot discover many obstructions and disorders of a smaller degree. The chest may be immobile in parts, but whether from blocked-up air tubes, diseased lung, liquid or air in the pleura, or any other of the various causes, sight and touch will not inform us.

We are driven, then, to try another sense which may reach beyond the surface,—the sense of HEARING. Now this sense is not generally cultivated as our sense of vision is. "Seeing is believing:" to see a thing is almost the same as to understand it. The same can scarcely be said of hearing; but yet, in cases where we have exercised this sense, we by experience render its indications nearly as instructive as those of sight. Thus we know the different sounds of carriages and horses in the streets, the foot-steps of walkers, the sounds of rain falling, of wind whistling, and so forth; and these several noises are the acoustic signs of the nature or physical character of the various bodies that produce them. So also the acoustic properties of the chest and its organs, the sounds that are produced in them or elicited from them, may by study instruct us as to the nature and condition of the matter of which they are composed.

Now there are two methods of studying these phenomena:—1. By mere individual experience: just as the infant studies the form and appearance of objects by sight and touch, and in time becomes acquainted with them. 2. Through a generalization of that experience in the laws according to which the phenomena occur. Personal experience is of course indispensably necessary to familiarize us with the character of the phenomena; but before it can inform us of their true nature and causes, experience must be extensive, and detailed to a degree that falls to the lot of very few; and even when thus obtained, its results, without generalization, must be so unwieldy and burdensome to the memory, that still fewer could duly profit by it.

We must avail ourselves of both methods. We must accustom our ears to the sounds in all their varieties, that we may be able by experience to know and distinguish them: but to understand their import, and to read the interpretation which they give of the condition of the parts that produces them, we must study them through the laws of which they are examples. We must consider what sound is; how it may be produced, transmitted, and modified; how the contents of the chest may produce it, and when produced can change it; and by comparing its general properties with the mechanism of the chest and its organs, we shall be prepared to

understand and arrange the phenomena that experience has discovered, or may hereafter reveal to us. By thus learning the acoustic relations of the chest, not merely as isolated facts, but as parts of an applied science, we may be enabled to escape in great measure the errors into which unintelligible matters of memory might continually lead us, and we shall be acquiring a rational pathology instead of resting on an empirical diagnosis. In this study, as well as in many of the subsequent parts of the course, you will find my little work on the *Pathology and Diagnosis of Diseases of the Chest* useful as a text-book ; and supposing that to be in your hands, I shall dwell less fully on the matters which are contained in it. In fact, there is so much new matter to be brought before you, or at least so much new application and arrangement of that which is already known, that I shall often refer you to the best works for common details, and give our time chiefly to the development and application of principles which are not, or but imperfectly, to be studied elsewhere.

We must devote the next lecture to a subject purely physical, the nature and properties of SOUND.

LECTURE V.

On the Nature and Properties of Sound—Sound a kind of Motion—Explanation of Vibrations of Sound—Vibrations of Cords, of Solids, Liquids, and Air—Effect of Sound on the Molecules of Bodies—Explanation of Duration of Sound—of Pitch, Harmony, and Discord—Conduction of Sound—Reflection of Sound—Modified Echoes—Sources of Sound—Modes of Increasing and Transferring Sound—Sounding-boards—Ear Trumpets—Difference between the Reflections of Sound and Light.

Now, gentlemen, to supply us with what we found needful in the last lecture, I am going to give you a brief explanation of the nature and properties of Sound. Perhaps you will be disappointed at finding that I do not propose to exhibit to you the various kinds of apparatus and instruments, by which lectures on acoustics are made attractive, if not instructing. I purposely avoid every illustration but those of the simplest and commonest kind ; and if I can judge by my own experience, you will learn more of the nature and laws of sound by reflection on facts and phenomena that are always within your reach, than by the most striking and wonderful displays wrought by the complete apparatus of the lecturer.

Let us begin with the abstract question, what is *sound*? *Sound* is not a matter, a separate thing ; it is a condition of matter. It is a *particular kind of motion* in matter ; and this motion being communicated to the matter in contact with our organs of hearing, produces that physiological impression which we call sound. This same motion, when examined by another sense, as sight or touch,

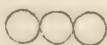
can often be seen or felt to be motion. Thus, when I strike a cord of this guitar, I not only hear the sound, but see and feel it move. We shall find hereafter many cases in which we can feel the motions of sound; and in all other instances where we hear sound, we know there must be motion. Our organs of hearing are more sensitive of this motion than our eyes or our fingers; they can feel finer degrees of it, because they are specially adapted to it.

But what kind of motion is this which constitutes sound? It is not a slowness of motion; for, as you perceive when I move this stick slowly through the air, it gives no sound. Neither will mere velocity of motion suffice; for, as you know, the earth is moving with great velocity, "without a whisper in its silent course." Now observe, by an example, what sort of motion produces sound. When I move this stick against something which offers resistance to it, as when it strikes this table, it gives sound; or when I move it so quickly through the air that the air resists it, we then have sound. Besides *motion*, then, we must have *resistance*; and this is so essential an element of sound, that I have ventured to define sound to be *resisted motion*, or more exactly, *motion of a certain force resisted by a certain force*. The moving force, and the resisting force, impelling the matter alternately to and fro, in opposite ways, constitute what are called the *vibrations* of sound.

This word *vibration* is a very good one for us, because it describes the nature of sound better than the words *wave*, *undulation*, *pulse*, &c., which rather imply the relations of sound to distance, and are less simple in their signification. Now if you want to understand what vibrations are, look at the bass cord of this guitar; it is now slack, and when I touch it you can follow, and almost count, its to-and-fro motions with your eye; these motions give no sound, or only a slight flutter, which is accidental. But observe how, as I gradually tighten the cord, its motions become quicker; and now you hear a sound. Now the silent vibrations which you could follow with your eye are precisely of the same kind as those which produce sound, except that they are slower; so are the vibrations of a pendulum; and as we can watch these, let us study in them the nature of vibrations in general. When I move this pendulum out of the perpendicular, and then let it go, you see it not only falls back to the perpendicular, but its momentum carries it beyond, to describe part of a circle on the opposite side, from whence it again sways back, past the perpendicular, to the side from which it started; and so on, backwards and forwards, for some time. Now the cause of these vibrations are two opposite forces, which alternately predominate—the disturbing motion or momentum, and gravitation. So, also, in the vibrations of a cord or wire fixed at both ends, the opposite forces are the disturbing force or momentum, and the elasticity of the cord; the first carrying the cord out of a straight line, and the latter tending to restore it to it. But I must not dwell too long on elements that may be known to you already; or that may, at all events, be mastered by a little reflection.

You will perhaps say, this is all plain enough in the case of a pendulum or a cord, but how can solid bodies, or liquids, or air, vibrate? Where are the contending forces here? I answer to this, just reflect a minute on the intimate constitution of these bodies. All matter is composed of molecules, which are held in their places by certain mutual forces of attraction and repulsion, which resist any forces tending to displace them. And now mark me, this resistance is not absolute, but of the same kind as the resistance which these guitar cords offer to my fingers; they yield for the moment, but spring back again, and vibrate to and fro until they recover their state of rest. It is an *elastic* resistance; and it is this property which constitutes in bodies what is called *molecular elasticity*. I will illustrate this by a diagram that will very well represent the vibrations of molecules.

Suppose the molecules of matter to be elastic spheres or spheroids (which, if not in themselves, yet, in their spheres of attraction and repulsion, they certainly are), we may represent them thus—



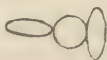
If an impulse be communicated to the first of these elastic spheres, the first effect will be to flatten it thus—



But its elasticity causes a reaction, which not only restores its shape, and communicates the impulse and flattening to the next molecule, thus—



but springs beyond it into an oval of the opposite direction, the original impulse having been then transmitted to a further molecule, thus—

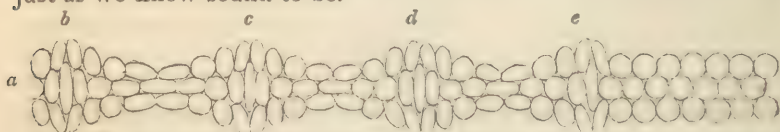


There is then another reaction, which throws it beyond its spherical into its first oval shape; and so on until the equilibrium of forces is restored, a molecule is rapidly undergoing all these changes—



Now this very diagram, which represents the changes which constitute the vibrations of a single molecule in a certain period of time, will also serve to show how an impulse, and the vibrations

which it causes, are propagated through distance, along a series of molecules ; this is *propagation*, or *conduction of sound*. You will now, too, perceive what is meant by a *wave* or *pulse* of sound, which I just now said is a more complicated thing than a vibration. It is, in fact, a series of molecules in the different stages of a vibration. Thus, in the diagram above, every five molecules contain a wave, the last being in the same condition as the first, and being ready to go through the same series of changes again. As I have not time for much explanation, I have constructed the diagram below, which exhibits more fully these waves, and the changes that constitute them. You are not to suppose that it is mathematically exact, but it exhibits very well the principle of the phenomenon ; and if it sets you a-thinking, you will, making allowances for the necessary corrections, get from it a better notion of the nature and phenomena of sound, than from any of the usual illustrations. It represents the progress of an impulse, followed by its opposite reaction, through a triple row of spherical molecules, which are seen beyond *e* in their state of rest ; the impulse, which began at *a*, not having reached them. The successive waves or pulses, *d*, *c*, *b*, are the results of the alternation of the impulse and resisting, or reacting forces, before explained ; and you see how an impulse, impressed on a single molecule (*a*), is communicated, laterally as well as in a direct line, just as we know sound to be.



By substituting elliptical molecules, we might represent the sonorous properties of those bodies that have a polarity, or whose density is greater in one direction than in another, as in the case of wood.

Well, then, having seen what sort of a motion that of sound is, and how it spreads itself through bodies, let us bestow a few minutes on the differences of quality of sound. Why does this piece of metal give a long ringing note, whilst this piece of wood, when struck in the same way, yields only a short knock ? It is because there is a greater equality in the molecular elasticity of the metal, so that the molecules continue to vibrate without interfering with each other. Let us illustrate this by the pendulum. Here are two pendula ; and you see, when I set them in motion, they swing harmoniously together, without interfering with each other, because they are of the same length, and therefore vibrate in the same time. Now I shorten one of them a little : now you see their times of vibration do not correspond, and they soon clash and stop each other's motions. It is just the same with the molecules of different bodies. Where their elasticity is uniform throughout the mass, the molecules vibrate in the same time ; and not interfering with each other, the vibrations are continued, and constitute a note

or tone, as in a simple piece of metal or glass. The molecular elasticity of wood, on the other hand, is not uniform; some molecules vibrate quicker than others, and the vibrations are consequently soon stopped. In softer bodies the molecular elasticity is still less uniform; their vibrations are therefore still shorter, and less perfect; they scarcely yield any sound; and they can choke the vibrations of other bodies with which they are in contact. Thus, see how little sound I get by striking this cloth; and when I bring this tuning-fork, which is sounding, in contact with it, the vibrations are immediately stopped. Yet we can get a sound out of this cloth; and remark how: by stretching it until it is tight, which increases and renders more uniform the elasticity of its molecules. Observe the same thing in this piece of membrane: whilst it is loose and flaccid, it scarcely gives any sound; but when I stretch it on this ring, it becomes quite a little drum. You see, then, why some bodies are sonorous, and others not.

Let us take up another difference, that of *note*, or *pitch*. Why does this short piece of metal, when struck, give a high sharp sound, whilst this long bar gives a low bass note? Because its vibrations are quicker; and it is an ultimate fact, that the sensations of shrillness, or deepness, in sound, depend on the number of the vibrations in a given time. You shall see the proof of this. Here is a long steel wire, fixed at one end: it now vibrates so slowly, that you can count the vibrations; but it produces no sound. I shorten it, it vibrates more quickly; and now it gives a low tone. I shorten it more; you see it vibrates still more quickly, and the tone is raised. I now fasten it much shorter; you hear how sharp the tone is; but the motions are so rapid, that you can scarcely see them. Observe the same corresponding changes of phenomena as I loosen or tighten the cord of this guitar. Again, when I blow into this short tube, you have a shrill note; with this long one the note is deep. You cannot see the vibrations here, because the vibrating body is air; but you can easily understand that a short column of air will move more quickly than a long one; and when I blow hard into the long one, I get a note an octave or more above, because the force of the blast increases the elasticity of the column, which therefore doubles its vibrations. I could add many amusing illustrations, but our time allows me to add only a word or two on a subject immediately connected with this—harmony and discord. When two sounds strike the ear at the same time, their vibrations will either combine or not, according as their numbers correspond or not in some simple arithmetical ratio. Thus, when a sound of 40 vibrations in a second strikes the ear at the same time with another of 40, the sounds will combine, and form the harmony of *unison*. Again, when one of 40 occurs at the same time with one of 80, the vibrations unite in the regular proportion of one to two, and the harmony of the *octave* results. So also with concords of the *third*, the *fourth*, the *fifth*, &c., there is a simple arithmetical relation by which their vibrations can unite; but when sounds

of such numbers as 40 and 45, 40 and 53, and the like, occur together, their vibrations cannot coalesce, but, affecting the ear with conflicting motions, produce the impression called *discord*.

Conduction of sound is the transmission of sonorous vibrations through bodies: and this obviously depends on the same properties, of strength and uniformity of molecular elasticity, which render bodies sonorous; and I shall merely illustrate this by an experiment. This tuning-fork, when struck, gives very little sound as long as it is held between the fingers; but when its vibrations reach the sounding-board of this guitar, its note becomes loud and clear. Now, by placing different bodies as means of communication between the tuning-fork and the guitar, we may judge comparatively of their conducting power. Through this long deal rod you perceive the sound is conducted very well; the longitudinal fibres of this wood are very rigid and uniform, and its lightness makes it easily receive the vibrations of contiguous bodies. This iron rod, too, answers well, for its molecules are strongly and uniformly elastic, and resemble those of the tuning-fork, in which the vibrations begin. But observe, when I substitute this handkerchief, or this sponge, although the distance is much less, the sound is not transmitted through them. When I draw the handkerchief tight, you can then just hear the sound feebly transmitted. Rigidity, or uniformity of tension, then, and similarity in density, or lightness of mass, are the qualities which render bodies good conductors of sound; and the opposite qualities make them bad conductors, which either do not receive the vibrations, or, receiving them, promptly choke them.

I have just said that some bodies do not receive the vibrations from other bodies; neither do they destroy them. What becomes of them, then? They are *reflected* back into the media from which they come. Now *reflection of sound* is an important phenomenon, and we must look into it a little. Suppose some light body—such as a cork ball, or an air-bladder—to strike a hard heavy body, such as a ball of metal; it would communicate little or none of its motion to the metal ball, but would rebound from it—that is, its motion would be thrown back upon itself. It is just so with the motions of sound. When the molecules of a light body, such as air, vibrate in contact with a hard body of much greater density, such as a stone wall, the motions are scarcely communicated to this body, but are reflected back; and this the more completely as the contrast between the density of the two bodies is greater. Now such a reflection of sound at a distance, you know, constitutes what is called an echo: here the reflected sound arrives at the ear later than the original sound, by the time which it has taken to travel to the reflecting surface and back. Sound travels through air at the rate of 1130 feet in a second; so you can easily perceive that at short distances, unless the reflections be repeated several times, the reflected sound will not be distinct from the original sound, and there will be no echo. Now in large rooms, particularly when empty,

the reflections are repeated many times, and you therefore hear a reverberation more or less continued, and generally altered by the reflection : but it is in small chambers, or cavities, of from six or eight feet down to two or three inches in diameter, that echoes present the most remarkable modification of a ringing or tinkling kind. This is what you may hear in empty barrels, bottles, and other hollow objects. When any sound or impulse reaches the air within them, it is reflected from side to side so rapidly that the number of reflections becomes as the number of vibrations, and gives a note the pitch of which is determined by the relation which the diameter of the cavity bears to the velocity at which sound travels through air. This subject requires a little consideration to understand it; but you will find in it the explanation of several phenomena intimately connected with the subjects to come before us. We have no time to dwell on it now.

We have seen that a certain kind of resisted motion constitutes sound: we have now to run rapidly over a few instances of this, in various modes of producing sound. The most familiar modes of producing sounds in solids are by percussion, collision, and friction. The two latter are, in fact, modifications of percussion, consisting of repeated percussions of the particles of the surfaces or edges of bodies. When these percussions are repeated with a certain quickness and regularity, they constitute a continued note. This cord is wound round with wire which forms a series of rings on it: when I rub the edges of this card slowly along it, it produces a *click* at each ring, and you only hear a succession of clicks; but when I pass the card rapidly, you do not hear the click, but a continued note, and this note is higher the quicker I move the card. Such sounds I call *click-notes*. They are not very musical, but they are interesting, as they present us with an analysis of many common sounds. The notes of the cords and wires of musical instruments, and of the parchment of the drum, need no comment: they are the result of the vibration of bodies rendered uniformly and freely elastic by artificial tension. Sounds depending on the vibration of air are instanced in explosions, and in the notes of the flute, pan-pipe, whistle, &c.. These are most readily conducted by air. Many sounds result from the combined motions of solids and air; the solid opposing a vibrating resistance to the passage of a current of air, and communicating its vibrations to the current. Such are the notes of reed instruments, trumpets, the *œlina*, &c.; and I believe that the human voice may be classed among them. Observe what a variety of notes I can get by blowing through this tube, with a thin piece of India-rubber stretched over the end of it. These are the same kinds of notes as those of the human voice; rather a cracked one, I admit, yet I have heard worse notes sung, and you will see the instrument is quite a rough and extemporaneous one. By drawing the India-rubber tighter, the tone is raised at pleasure. I have no doubt that we might, by a little coaxing, make the note more mellow; and it requires only a little mechanism to divide

this note into articulate sounds. But I leave it for others to make this artificial voice speak ; it has said enough for us.

There are certain modes of increasing and transferring sound, which we must just advert to. When I strike this tuning-fork, and hold its vibrating end at the mouth hole of this flute, it produces no sound unless I stop up some of the finger-holes, so as to produce the fingering of a note in unison, or in close harmony, with that of the tuning-fork ; and then observe what a clear note comes forth. The column of air in the instrument so adjusted, has just that degree of elasticity that enables it to vibrate in unison with the tuning-fork, the notes of which it reciprocates. A cord tuned in unison will also reciprocate in like manner. Sounding-boards of stringed instruments are constructed with hollows of varying depth, that the air within them may reciprocate the different notes of the cords ; but the chief use of sounding-boards is to assist in transferring the vibrations of the solid wire, or cord, to a large surface of the air, which is so much more rare and yielding that it is but little impressed by the motions of so small a body as the cord alone. You have already seen this illustrated with the tuning-fork, and the sounding-board of the guitar ; but now you may perceive that this board, which has no hollow, answers in a less degree. The knowledge of these facts enabled Mr. Wheatstone, some years ago, to astonish the public with his *enchanted lyre* ; which was merely a sounding-board, communicating by a small rod of wood, or metal, with the sounding-board of a pianoforte concealed in another room.

Sounds produced in air may be directed and concentrated by reflecting surfaces. It is on this principle that ear-trumpets and speaking-trumpets are constructed. The former ought to be so made as to receive the greatest possible body of vibrating particles, and to concentrate them by as few reflections and turns as possible. If you go into the shops, you will find very few instruments in which these points are attended to. The greater number render the speaker's voice noisy enough, but they sadly confound his words. A large cone of paste-board, with a short curved tube from its apex for the ear, makes as good an ear-trumpet as can be constructed ; and we only want a little mechanical ingenuity to contrive one of this shape that shall be also portable, to bring it into general use. One word more on reflection of sound, which is applicable to this subject. It is generally said that the reflection of sound from a plane surface takes place like that of light, at an angle equal to that in which it fell on it. This is true with regard to sounds at some distance ; but when a sonorous pulse of air approaches a reflecting surface, by compressing the layer of air next the surface, it increases the elasticity of that layer, so that it begins to be reflected, or rather *refracted*, before it reaches the reflecting solid. Its course, therefore, instead of being like that of light, thus —



is turned in a course thus —



This is the reason why it is not necessary to construct instruments for concentrating sound by reflection, with the nicety that is indispensable in those for reflecting light ; and it is by a similar property that close tubes of air have the power of conducting sound so much further than open ones. But we must conclude this subject, and in the next lecture take up the acoustics of the chest.

If you wish to study sound further—and you would find it both amusing and instructive to do so—I should advise you to consult Sir J. Herschell's Essay, in the *Encyclopædia Metropolitana*, and Dr. Arnott's *Elements of Physics*. These works do not contain all that I have been giving you ; for having a new application of acoustics to deal with, I have found it necessary to investigate properties of sound which had not been fully studied before. This investigation has been so far successful, as to enable me to reduce to simple principles all the acoustic phenomena which we have to consider ; and I now hope that these principles will prove as intelligible and instructive to you as they have been to me.

LECTURE VI.

Physical Examination of the Chest (continued)—Acoustic properties of the Chest tested by Percussion—Causes of the sound of Percussion—Modes of Percussion ; immediate and mediate—Varieties of the Sound in different Regions ; in different states of Respiration—Practice of Percussion—Acoustic properties of the Lungs, manifested by the motions of Respiration—Sounds of Respiration, Inspiration, Expiration—Tracheal, Bronchial, and Vesicular Sounds—Varieties of the Sounds ; Puerile Respiration, &c.—Duration of the sound of Respiration, &c.

WE have now, then, to consider the *acoustic* properties of the chest and its organs. We have explored these parts in their relations to sight and touch, and we have next to examine them in relation to sound, which we now know to be merely a kind of resisted motion.

Well, as we tried the sonorous properties of metal and of wood, by striking them, and observing the character of the resulting sound, so we strike the chest to judge of the nature and condition of its materials. The practice of percussion, as a mode of diagnosis, we owe to Avenbrugger. We will first inquire into the principles of this process. You hear that the chest, when struck abruptly with the ends of the fingers, gives a rather deep, and not very short sound ; which implies that the vibrations are not quick, and that

they do not instantly cease. When I strike in the same manner on the thigh, there is a very different sound—a short dull tap, implying that the vibrations do not continue. I get the same dead tap when I strike that part of the chest in which the liver lies ; but you may observe, by referring to the diagrams, that those parts of the chest under which the lungs are, all yield more or less of the deep hollow sound.

Let us examine a little more into the seat of this sound. Is it in the air, or in the solids of the chest ? We will first try the properties of sounds produced in the air of hollow bodies. Here is an open India rubber bottle, which gives, when I tap on it, a hollow sound. But if I close its mouth with my finger, you hear how the sound is changed. Try this with the chest : I close the glottis whilst I strike my chest ; it does not sensibly alter the note. Again, the sound in the air of hollow bodies is deep in proportion to their size : remark how much deeper the tone of this large bottle is than that of the little one. Try this with the chest : by enlarging and diminishing its hollow by inspiration and expiration the sound on percussion remains much the same, except that the extremes of inspiration and expiration slightly raise it ; whereas, were the sound produced in the air within, the increase of this air should uniformly lower the sound, and the diminution raise it.

As it is not, then, the air that sounds on percussion, it must be the solids ; and observe how the solid construction of the chest favours its vibrations. Here are a set of membranes, thin muscles, and integuments, strained on an elastic framework of bone and cartilage, and free to vibrate so long as the organs within do not check their motions. Let us find something familiar to illustrate this ; my hat will do very well. The crown of this hat is free to vibrate, and gives a good hollow sound when struck. I put into it this handkerchief, which when loose, may serve to represent the air-filled tissue of the lung. It still sounds well, because the handkerchief does not check the vibrations. But when I bring my hand in contact with the crown inside, and then strike on the outside, you perceive the sound is rendered short and dull, because the vibrations are stopped by the solid within. *Suppose* I were to fill my hat with water (for I am not going to *do* it ; I wont try its *water-proof* so far) ; this, too, would check the vibrations, and you would have no sound on percussion but a short dead tap.

It is just so with the chest. It is in itself more or less free to vibrate ; but it receives the character of its vibrations from the organs underneath, which return them, or check them, according to the density of these organs. Thus you find, where the lungs lie, the chest sounds deep and clear ; but here, below the sixth rib on the right side, where we come over the liver, or here, to the left of the lower part of the sternum, over the heart, the sound is short and dull, the vibrations being checked by these soft solid organs beneath. So also you can at once perceive that morbid changes of the organs,

such as condensation of the lung by disease, or the pouring out of serum into the pleura, would in a similar way arrest the vibrations, and render the sound dull in those parts of the chest where these changes occur. On the other hand, suppose changes of an opposite kind to take place, such as dilatation of the air-cells of the lung, or an effusion of air into the pleural sac, the walls of the chest will then have under them an increased spring, which will make them vibrate on percussion even more strongly than usual, and yield a clearer sound.

These illustrations are enough to show the general principles of the acoustic examination of the chest by percussion. It is a test of the density and elasticity of the materials within the chest : as diseases alter these qualities, so will they alter the sound on percussion, which may thus announce their presence. A few more considerations will help us to some useful practical applications of these principles.

As we have seen that the walls of the chest give the sound which we hear on striking the chest, so it is plain that they must be sufficiently tense and elastic to vibrate on being struck. The chests of some persons are so loosely put together, and so flaccid, that they give but little sound, although the organs within are quite healthy. In others, again, there is such a mass of fat and loose integument on the chest, that the walls are completely muffled by it, and they sound but little on percussion. The same difficulty occurs in most cases in certain regions, where muscles of considerable thickness, or the *mammæ* in females, lie on the walls. In other cases, again, the walls of the chest are so drawn in by contracted adhesions, that they are not free to vibrate, and give a dull sound, although the lungs within them may be comparatively healthy. In all these cases we must give to the part struck the equal tension which is wanting, by pressing on it a small piece of some firmly elastic body, such as wood, ivory, stiff India rubber, or the like. You can get plenty of sound by striking this ; and if it be firmly applied to the chest, the density of the contents within will modify this sound, just as it modifies that of percussion of the naked walls of the chest. You perceive that the sound which I get on striking on this plate of ivory pressed on my chest, is the same in character as that which I get by striking the chest itself, only it is louder ; and as percussion on it gives no pain, I can strike with force sufficient to make the vibrations reach the interior, through any thickness of fat or muscle. By this means we can get at the sonorous qualities of the thoracic viscera through the scapulæ and muscles of the back, and through fat or oedematous integuments of any thickness. In this way, too, we can get the resonance, or sonorous quality, of any part of the abdomen. In some regions, as in the right iliac and left hypochondriac, the gaseous distension of the colon and stomach often stretches the solids over them enough to make these solids yield a drum-like sound when struck ; but the pressure of a solid plate on them will

always give these and other regions such a tension, that a stroke on its surface will be modified by what lies underneath.

We owe this method of *mediate* percussion to M. Piorry, who calls this percussion-plate a *pleximeter*; and it is so much better than immediate percussion, that it is now generally preferred. There is, however, an improvement on it, which is, I believe, due to an Englishman, Dr. Skerrett; this is, to substitute for a pleximeter the fingers of the left hand. I need say little to recommend this method of percussion; if you do not already know it, you will soon find out how convenient and *handy* it is, how nicely you can fit the fingers to the inequalities of the chest, sometimes singly, sometimes together; sometimes with their palmar surface outermost to strike on, but generally with this applied to the chest and their backs to strike on, and so forth, as I will show you at a future lecture, when I will give you practical demonstrations of all the different methods of examination.

Now that we know how to percuss the chest so as to make it sound, let us see how this sound in different parts ought to be affected by the organs within; and here, again, you see we have need of the topographical knowledge that I said was necessary for a correct physical examination of the chest. These diagrams will assist us; and for the different sounds on percussion in different regions, I must refer to you the table and plates at the end of my work on Diseases of the Chest.

You see here, coloured light pink, the shape of the lungs seen through the parietes; and over the whole of that you get, more or less, the resonant pulmonary sound on percussion. In all the upper parts of the chest, before, behind, and at the sides, this sound is pure and unmixed, and equal on both sides, because there is nothing but lung underneath, as you see represented in the drawing by the unmixed pink. But between the fourth and fifth rib on the right side, you see this pink begins to get a shade of purple from the liver, that here rises into the central part of the chest beyond the lung; and this purple tint becomes darker and darker as the lung is thinner, and the liver approaches the surface, until we come to the margin of the lungs, where the light pink of the lung quite ceases, and the unveiled purple of the liver denotes that this organ is in contact with the walls of the chest. Now, is there any mixture of the lung and liver *sounds* where we see this blending of their tints in the diagram? I say yes; and I will give you the proof of it when we have a living subject before us. As soon as we get down to the upper level of the liver there is a very slight deadening of the pulmonary sound on percussion; and this deadening increases down to the margin of the lungs, below which it is entirely changed into the dead hepatic sound. You see by this that the stroke of percussion reaches a considerable depth, to organs an inch or more from the walls; and whatever it reaches may modify its sound. This suggests to us, that by varying the *force* of the stroke, we may make the impulse of percussion reach to different depths, and derive the character of its sound from the superficial or the deep-seated organs, as

we will. So, after some practice, you will find that where the lung overlaps the liver, *strong* percussion will give you a shorter, deader sound, than *gentle* percussion. Strong percussion receives the character of its sound from the liver as well as the lung; whilst gentle percussion, as by *filliping* with the finger and thumb, does not pass beyond the thin layer of the lung, and gives still the pulmonary sound.

Let us look to the left side of the chest. Here, at the sternum, about its junction with the third rib, we see the light pink of the lungs taking a scarlet tinge from the heart, and you can see its shape blushing through the lungs down to between the fifth and sixth ribs, about an inch below and before the left nipple, where, from the brightness of the colour, you may see that the apex of the heart approaches very near the walls of the chest; and to the left of the lower third of the sternum, its body is in contact with them. So there is a mixture of dull and clear sounds on percussion in these situations, although from the movements of the heart and lungs, the spots where one *shades* into the other are not fixed. You see, in the lower part of the left side, a yellow tinge, which rises, in a faint degree, as high as the mammilla. This indicates the vicinity of the stomach, with its clear drum-like sound, which becomes mixed with the pulmonary sound in this region, but to a degree varying according to the quantity of air contained in the stomach. Here, again, is the spleen, which, as you see by this blue tint, affects the sound of the small portion of the lateral lobe of the left lung.

Now you must not forget that the motions of respiration may produce changes in the character and relative positions of some of these sounds. Inspiration, as it enlarges the lung, renders the pulmonary sound clearer, and extends it over every part of the heart, and over a considerable portion of the liver. As we found that the complete and equal enlargement and contraction of the chest, as seen and felt, are signs of the free condition of the respiratory organs, so the sound on percussion becomes an additional sign of the healthy action in proportion as the clear pulmonary sound is extended at each expansion of the chest. Observe, when after a full expiration I strike my chest over the region of the heart, and over the middle region of the liver, the sound is dull, and scarcely pulmonary. I take in a full breath; and now you hear how clear it sounds all over these regions. Percussion is a test, therefore, not only of the *statical* condition of the lung, but of the *dynamical* state also. This point is not enough attended to by auscultators, and yet the neglect of it not only would deprive us of additional signs, but would tend to render deceptive the results of statical percussion. For example, in judging of the goodness of the sound on percussion, we generally compare the sounds on the two sides of the chest, or in two parts of the same side where naturally the structure and sounds are the same: but if we do not attend to the movements of respiration, we may strike one part when the chest is contracted, and the other when it is full, and obtain results which differ from this cause only, and not from any internal change.

In practising comparative percussion, therefore, in cases requiring delicacy, you should desire the patient to hold his breath for an instant whilst the comparison is made, and it is often useful to try the sounds when the chest is expanded to its utmost, when it is contracted, and in the intermediate states.

It would be going out of my order to describe the varieties in percussion from special diseases, but an illustration or two more will enliven the subject.

The indurations of the upper lobes of the lung in the early stages of phthisis are often small, and so scattered through its substance, that they scarcely affect the sound on percussion; but by a full expiration they are brought closer together, and if more on one side than the other, they may then more sensibly deaden the sound on that side, especially if gentle mediate percussion be used below the clavicles, and not on a very small surface. Again, the indurations, especially if of some standing, tend to restrain the lung from its full expansion, and if there be a difference on the two sides, it thus may be detected only on a full inspiration. In the disease called emphysema of the lungs, the air-cells are permanently dilated; they contain an unusual quantity of air, which expiration cannot expel; this may be detected by percussion as a dynamical test; the regions of the heart and upper part of the liver, being covered by the permanently distended lung, even after expiration, give a clear sound. There is one point more to notice respecting percussion at the extremes of the respiratory act. Full inspiration makes the sound clearer; full expiration, the contrary: but they both raise the tone a little; they render its pitch higher. Why is this? For the simplest reason in the world; both actions *tighten the drum*; they strain walls of the chest, and render their vibrations quicker, and therefore the sound higher. It requires a musical ear to detect these differences, and I do not think it useful to dwell further on them.

I think I need add no more on the principles of percussion; but to those who have not practised it, I would say, do so without delay, and on every opportunity; for, like every other art in which our senses are exercised, there must be practice to familiarize you with the phenomena, and the mode of obtaining them. I trust that the principles which I have given you will guide and assist you in this practice. There is no better way of trying to get this familiarity than by percussing your own chests. A few minutes when you rise in the morning occasionally spent in studying before the glass the outward marks and different sounds of the regions and their corresponding organs, will go far to instruct you in the practice of percussion, and in the character of the sounds in health; and the awkwardness and annoyance inseparable from the first attempts will be felt only by the person who is least likely to complain of them.

You will soon find even in these trials that some art is required even in mediate percussion, which is the easiest. The finger or fingers of the left hand should be closely pressed on the walls of the chest, and if the object be comparison of the two sides, they

should be placed on corresponding parts, whether between the ribs, along them, or across them. Take care, too, that the mode of striking be the same, whether you strike with one or several fingers, with their tops (in which case you must keep your nails pretty closely cut), or the flat of the last phalanx, or with the knuckles; each of which modes is sometimes preferable. I often find *filliping* with the middle finger and thumb give more uniform and delicate results: especially when the patient is in an inconvenient position, or suffers from tenderness of the walls of the chest, this is the best mode for abdominal percussion. In doubtful cases, however, various modes must be tried and practised, as I have before remarked, with due relation to the period of respiratory movements. There are a few cases in which a little plate of thin wood or ivory, as recommended by M. Piorry, may be used with advantage; it should be covered with soft leather to prevent the clack of the fingers on its surface. In M. Piorry's works "*De la Percussion Mediate*," and "*Du Procédé Opératoire*," &c. you will find some useful descriptions of the varieties of sound obtained by this instrument, but he much exaggerates its advantages. If you wish to know more of the principles of percussion, you may consult a paper of mine, published in the Medical Gazette of January, 1837.

Now let us inquire into other modes of producing sounds in the chest, which may prove acoustic signs of the condition of the organs within. There are the motions of respiration, will not they produce sound? The contractions and relaxations of the muscles of respiration are in general too gentle to cause sound; but when forcible or sudden, they will sometimes occasion a sound of tightening, of the kind called muscular sound. This is of no consequence, and gives no signs. But there are the internal motions, and the attendant passage of air to and fro in the lungs; these produce sounds, and inasmuch as this passage of the air is the great object of respiration, we may expect to find in these sounds signs of the manner in which this object is accomplished. These, and most of the other acoustic phenomena of the motions of the chest, were first discovered and described by Laennec, who may well be considered the father of the art of auscultation. We shall endeavour so to illustrate and extend this art, by the aid of physical and physiological science, that we may be enabled to deduce respectively from the phenomena the condition of the organs, and from any known condition of the organs, the phenomena which it would produce.

The air enters the lungs by atmospheric pressure, to fill the increased space made in the chest by the action of the muscles of inspiration. On its way to the most expansible parts of the lung, the fine tubes and cells, it strikes against the sides and angles of the trachea and its ramifications, with force sufficient to produce a particular hollow blowing sound. You may hear this on applying the ear to the fore part of the neck, or at the top of the sternum. As the current of air becomes subdivided and spread in the small

bronchi, it loses a part of its velocity, and the sound becomes of a more diffused and less hollow character ; it is more like the sighing of a gentle breeze among the leaves of trees, and in passing into the cellular terminations, all of the hollow or tubular sound is lost, as you may find on applying your ear to most parts of the chest. This sound may partly depend also on the opening and stretching of the tubes and cells. Where inspiration ceases, expiration begins, and a portion of air is pressed out of the cells and small tubes by the collapse of the walls of the chest, and by the contracting properties of the pulmonary tissues. Now, remark the difference between inspiration and expiration. In inspiration the air is the moving body, and rushing through the tubes distends the passive lung. In expiration the lung is the moving body, and by its contraction (backed by external pressure) drives before it the passive air. In either case there is a pressure exerted between the air and the interior of the cells, and doubtless this proves the means of assisting the chemical changes that take place. But you can see that there must be a difference between the sounds of inspiration and expiration. In inspiration, air moving with some velocity meets with the resistance of the angles and sides of the tubes and cells which it has to dilate. Here must be sound in the whole passages of the air, from the nostrils down to the pulmonary cells. In expiration, the motion begins with the lungs, and, the air passively yielding to it, there is not motion or resistance enough to produce sound, until by the converging together of the small tubes, the impelled air is gathered into a current in the larger tubes, where, impinging against their sides with its now acquired velocity, it at length produces sound. These remarks will enable you to perceive, that in natural respiration there are three kinds of sound—*tracheal*, *bronchial*, and *vesicular*. In expiration, at most only two, *bronchial* and *tracheal*. I am not sure who first remarked these differences, which were overlooked by Laennec. They are mentioned by Andral, Louis, and Dr. Cowan, who accredits them to the late Dr. Jackson, a young American student at Paris, whom I knew as an uncommonly zealous and clever observer. They are worth notice, and are plainly produced in the manner which I have explained. We shall say no more at present of the distinctions between these parts of the act of breathing, but class them generally, as Laennec did, as the sounds of respiration.

I have said that the sounds of respiration can be heard on applying the ear to the chest ; they are transmitted through the parietes with sufficient distinctness from the parts underneath ; and as the healthy sounds vary in these different parts, we may judge of the natural distribution of the tubes by listening to these sounds. Thus we find in any part of the neck, and at the upper part of the sternum, there is the hollow blowing sound which results from the passage of air to and fro in the trachea, which is therefore called *tracheal* respiration. A little lower down than this, over the space of two or three inches on each side of the top of the sternum,

between the scapulæ, and sometimes in the axillæ, there is the sound called *bronchial* respiration, because its whiffing or tubular character denotes that the sound is produced by the passage of air in the bronchial tubes. Then there is the *vesicular* respiration, which is heard in most other parts of the chest; it is a diffused murmur, caused by the air penetrating through the minutest tubes, and into their numerous vesicles or cells.

Now a question will occur to you (at least, I hope it will, that you may be more interested in the answer), how is it that the *bronchial* respiration is heard in comparatively few parts of the chest, when we find bronchial tubes of considerable size in most parts of the lungs, to within an inch or so of their surface? Why is not the sound a mixture of the tubular and vesicular sounds? Just consider that the sound in these tubes must be conducted through the tissue of the lung to its surface before it can reach the ear; and what sort of a conductor of sound will this tissue be? We found that equality of density or rigidity renders a body a good conductor of sound; but here we have a flaccid tissue, composed of the unequal materials, membrane and air; and sound in traversing an inch of this tissue would have to pass from air to membrane, and membrane to air, thirty or forty times. We saw how a loosely folded cloth stops sound; still more especially does this spongy tissue of the lung arrest all the slighter sounds that are produced in the tubes within it; so that what we hear outside the chest, unless the tubes underneath are of very large size, or very near the surface, is only the vesicular sound of the superficial parts of lung. You may, if you please, take a hint from this, if you want to shut out the noise of a street, or of a noisy neighbour, or if you wish to keep your own noises to yourself, you will have on the shutters, or in the walls, some loose porous body, such as wool, cotton, shavings, or the like, which will effectually arrest and choke the vibrations in their passage, and will in a measure isolate you with regard to sounds.

And now mark an important corollary to our last proposition. As this arrest of the sounds of the interior depends on the light spongy structure of the lung, so any disease increasing the density of that structure will increase its conducting power, and enable it to transmit the sounds. Hence we find that a great increase of solid or liquid in the lung, as in pneumonia or tuberculous deposit, or the compression of its superficial parts by a moderate quantity of liquid in the pleura, as in a recent pleurisy, will often not only diminish the vesicular murmur in consequence of the obstructed state of the cells, but will add also a bronchial or tubular sound of breathing in those parts where naturally the respiration is purely vesicular.

Now, I think, we understand the ordinary sounds of respiration; let us attend to some varieties of them. As these sounds depend on the resisted motion of the air, so they will vary according to the velocity of that motion, and the degree of resistance to it; they will

be loud when the air passes in and out forcibly and quickly, and low when it passes gently and slowly. So when you are listening to a person's breathing, it may be scarcely audible at its ordinary rate, but if he breathe quick and short, it will be distinct enough. Taking a *long* breath may not answer the same purpose, for although much air is thus taken in, it may not enter with sufficient rapidity to cause the increased sound. Coughing answers better, for the long inspiration which follows coughing is generally quick also; and it is often useful where the sounds are obscure, to magnify them by this more forcible act. But there is a limit to the power of increasing the sound of respiration by increased effort. If a person tries to breathe very hard and quick, as after violent exertion, the movements of the lungs cannot keep pace with those of the external muscles of respiration, and the air does not freely enter, the sound will be diminished rather than increased.

As we can vary the sound of respiration, by varying the act in the same individual, so we find that a difference exists naturally in different individuals: in some, as in many robust adults, you find the ordinary respiratory sound very low and faint; in others, as in children, in nervous females, and in slight irritable persons, you find the sound loud and distinct. In the last cases the respiratory movements are more brisk, and although air may often not be taken in more frequently, or in such great quantity as in the other cases, yet it enters more suddenly, and meets with greater resistance in its passage, so that it must cause more sound. As this loud respiration is commonly met with in children, Laennec called it *puerile* respiration. So, also, by rendering the respiration quicker and more energetic, you may make its sound loud in those cases where it is naturally faint, as by the quick short breathing which I have just mentioned; or better still, by desiring the person to hold his breath for a while; the quick strong inspiration which then follows is noisy enough. Disease will sometimes bring about the same change: thus if a considerable portion of the lungs be obstructed, the force of the act of breathing will be concentrated on the remaining portions, and the air will be carried in and out of them with unwonted energy and noise. Hence Andral terms this partially increased respiration *supplementary*. So also, under some circumstances, without any obstruction, the want of breath may be increased—as it happens during moderate exercise, and in some degree during the process of digestion, and on exposure to cold: here respiration is more energetic, and its sound louder. Further, as the act of breathing depends on a particular impression on the nervous system, so you can easily perceive when this system is preternaturally sensitive, the ordinary impression produces an increased effect; and here, again, the respiration becomes more energetic and noisy. This is the cause of the increased sound of respiration in certain fevers and other diseases where the nervous sensibility is exalted. Lastly, it is possible, by an external restraint of some parts of the chest, to render the sound of respiration louder

in other parts. Thus, if you inclose the abdomen and lower part of the chest in a tight belt (or you may meet with the experiment already prepared in the persons of tight-laced ladies), you will often find the sound of respiration in the upper parts unusually loud ; and you see by the heaving of these parts how their motions are increased.

Now you see there may be much variety in the sound of respiration, without disease of the lungs : and excepting in the case last mentioned, it is where there is a comparative difference in the different parts of the lungs, rather than any absolute difference, that disease of these organs is indicated. Thus, if you find the respiration loud on one side and obscure on the other, or clear in the lower part of the chest and indistinct in the upper, you may well suspect some obstruction to exist in those parts where the sound is obscure ; and the nature of that obstruction is then to be tested by percussion and other means.

There is another kind of variety in the respiratory sound that has not been attended to—a variety in its *duration*. In this, as in the other varieties, there are absolute differences in different individuals, and in the same individual under different circumstances ; but I shall only notice the comparative differences in the same subject at the same time, which alone constitute signs of disease. We sometimes hear the sound of inspiration on one side distinct and prolonged during the whole inspiratory act ; on the other side loud enough at first, but abruptly stopped before the act is complete, and often stopped with a sort of *hic*. This kind of hitch in the sound sometimes reminds me of M. Alexandre, the mimic ; imitating the noise of planing, he gives you the idea of meeting with a knot in the wood, by stopping short the noise with a *hic*. So the stop put to the expansion of the lung is often as sudden ; and this denotes that the obstruction is then complete. Hepatization of the lower portions of the lung will do this ; so will a moveable plug of tough mucus in the bronchial tubes. In other cases, again, we find the circumstances reversed ; there is in a part of the lung no sound during the first part of inspiration, but towards its end, when the chest is most expanded, there is a short wheeze. This happens where the bronchial tubes are so far obstructed that air will not pass through them until they are distended by a full inspiration, as in bronchitis. It happens also in pleuritic effusions, which distend the parietes of the chest beyond the medium state of respiration ; it is only the *acmé* of inspiration that can then introduce air into the compressed lung, and it is at this period alone that the sound is heard.

I must not detain you now with other morbid varieties of respiration, which will severally come under our notice when we consider the diseases which cause them. There is the *cavernous* respiration, caused by the passage of air in and out of an unnatural hollow or cavity in the lung ; and the *amphoric* respiration is where this cavity is so large, that the sound is like blowing into a phial, or into this India rubber bottle.

In the next lecture I will give you a tabular view of these and other varieties of respiration, and of their remarkable accompaniments, the various *rhonchi*, which are produced by an increased resistance to the passage of the air.

LECTURE VII.

Physical Examination of the Chest (continued)—Sounds of Respiration (continued)—The Rhonchi—Dry Rhonchi, Sibilant, Sonorous, Dry Mucous—Humid Rhonchi, Mucous, Cavernous, Submucous, Subcrepitant, Crepitant—Importance of the Rhonchi as Signs—Sounds of the Voice—Natural Sounds, their causes and regions—Morbid Sounds: Bronchophony, Ægophony, Pectoriloquy, Metallic and Amphoric Resonance, Pectoral Fremitus—Sound of Friction—Tabular View of the Sounds of the Organs of Respiration.

WE have hitherto considered the sounds produced by the passage of the air to and fro in the lungs, and we found that the varieties of these sounds depend on the size of the tubes and on the force with which the air strikes against their sides and angles, and that they may be shortened or stopped by various kinds of obstruction.

We have now to attend a little to a class of novel sounds which arise from *partial obstructions* to the passage of air,—obstructions which permit the air to pass, but not without such a resistance as causes an increased and modified sound. Thus, suppose a bronchial tube to be narrowed by the swelling of its membrane, or by mucus secreted by it, the air must pass through the narrowed portion with increased velocity and increased resistance, and hence the sound is changed from a simple breathing or blowing, to a louder wheezing, bubbling, whistling, or snoring, according to the nature of the obstruction. These new sounds Laennec called *râles*, or rattles: I have preferred the Latin term *rhonchus*, as more expressive, and this has been adopted by most English writers. I wish we had an English word that would do as well; for nothing injures the purity of a language more than the introduction of foreign words; and it is only pedantic to use them when English terms will express the same thing. And here, in relation to our subject, I enter my protest against the prevalent use of French words, in English speaking or writing, such as *bruit de soufflet*, *bruit de râpe*, *bruit de pôt fêlé*, *frottement*, *gargouillement*, *tintement métallique*, &c.; the simple translation of which would do just as well, and convey much more precise and intelligible ideas than these mystifying terms. We cannot, however, do well without the word *rhonchus*, or some other to be used conventionally in the same sense, as a generic term applicable to all those sounds which are produced by an increased resistance to the air moving through the lungs.

Let us attend to some of the species of *rhonchus*. I divide them into the *dry* and the *humid*, according to whether the impediments that produce them are solid or liquid. Of the dry *rhonchi* there is

the *sibilant* or *whistling rhonchus*, which is sufficiently described by its name, and may be generally imitated by whistling between the teeth. It is produced by the passage of air through a small and rather circular aperture ; and this aperture may be formed by a slight obstruction of a small tube, or by a greater obstruction in tubes of larger size. It generally occurs in tubes narrowed by swelling of their mucous and submucous coats, such as occurs in the early stage of acute bronchitis : but you have it also in asthma, where the tubes are congested and constricted by the spasmodic contraction of their circular fibres ; and you may have it also when viscid mucus clings to and diminishes the calibre of the tubes.

The *sonorous rhonchus* is a snoring, humming, or droning sound, and may vary in loudness and key from an acute note like that of a gnat, down to the grave tone of a violoncello or bassoon. It must be produced by obstruction leaving a flattened aperture, the lips of which, or the moisture on them, yield to the passing air with a vibrating resistance. Partial swelling of the sides of a tube, particularly at its bifurcation, a pellet of tough mucus in it, or external pressure on it, may cause such a flattened opening within the tube ; and the sound in question therefore occurs in various forms of bronchitis, and often accompanies tumours which press on the bronchial tubes. When caused by tough phlegm, coughing will generally change or remove it ; when from the other causes, it is generally more permanent. When quite permanent, it usually depends on the pressure of a tumour or some deposit outside the tube. The key of this note depends chiefly on the size of the aperture left ; when this is small the note is high, when large the note more of a bass : from this we may infer that the latter can only have its seat in the larger tubes ; but as a more considerable obstruction may flatten their calibre to the smallest size, these may also be the seat of the acute notes. You may imitate almost every variety of this rhonchus by blowing between the lips moistened with saliva and almost closed. This is the mode in which M. Alexandre imitated the buzzing of a fly.

There is another rhonchus which I call the *dry mucous*, because it is produced by a pellet of tough mucus obstructing a tube, and yielding to the air only in successive jerks, which cause a ticking sound, like that of a click wheel. When the air is driven very fast, these, as in the case of other click-sounds, pass into a continuous note, and constitute the sonorous rhonchus. Sometimes, again, particularly in inspiration, the click-sound suddenly stops ; the tough mucus being forced into a smaller tube which it completely closes, and may not be dislodged again, but by dint of forcible coughing.

Now, as any of these rhonchi may be produced in only one tube, and yet make a great deal of noise, you are not to suppose that they are important in proportion to the noise they make. It is rather when they are very permanent, or when several of them are heard at once in different parts of the lungs, that they bespeak disorder which may be serious, either from its permanency or its extent.

The *humid rhonchi* all depend on the passage of air, in bubbles,

through a liquid in the lungs, and their species are produced by differences in the size of the tubes, and the nature and quantity of the liquid, which cause varieties in the bubbling sound. Do not suppose I am trifling with your time because I ask your attention to some of these *bubbles*. Newton learnt much by studying the colour of a soap bubble, and he still left much in it for others to explain ; perchance, we may learn somewhat by studying the sound of the bubbles in the lungs ; at least we should lose nothing, as others do, by their *bursting*. A bubble is a portion of air contained and slightly compressed by a thin film of liquid, which preserves its continuity by its molecular or aggregative attraction : when this attraction is overcome, by the gravitation of the liquid, the motion of the air, or any other disturbing cause, the bubble bursts : as it bursts, the air from it, slightly expanding, gives to the adjacent air an impulse, which, if forcible enough, produces sound. In the bubbling passage of air through a liquid, the air is the moving body ; the liquid gives the resistance, and in proportion as these are strongly and suddenly opposed to each other, the louder will be the sound produced. If the air pass with force it will make most noise in a liquid of some tenacity which offers to it most resistance ; but if it move slowly, such tenacity may retard the breaking of the bubbles, and therefore diminish the sound. Again, air passing through liquid in large tubes will give most sound when the liquid is thin, because the bubbles form and burst quickly ; but in passing through very small tubes air will cause more sound with a rather viscid liquid, which, adhering to the tubes, is not carried before the air so readily as one of a thinner nature. Now, let us apply this rule to the bubbling sounds or rhonchi heard in the chest.

The *mucous rhonchus* may be heard in the large and smaller bronchi down to the size of a crow's quill, and in these different situations its gurgling or crackling presents different degrees of coarseness. It is an irregular and varying sound composed of unequal bubbles, and generally interspersed with some whistling, chirping, or hissing notes. Its most common cause is acute bronchitis, which after its onset is attended with a secretion of liquid mucus into the bronchial tubes ; and the passing of the sibilant and sonorous rhonchi of the first or dry stage into the bubbling of the second or secretory stage is often marked by a curious combination of chirruping and cooing notes, like those of birds in a bush. When the bronchial tubes become unnaturally enlarged by disease, or when morbid cavities are formed by the destruction of portions of lung, the bubbling of air through liquid in these, is of the coarsest kind ; it is quite *gurgling*, and if the liquid be scanty, has a hollow character, and is called the *cavernous rhonchus*.

When there is a little liquid in the smaller bronchi the bubbling or cracking is more regular, although the sound is weaker, and is sometimes only a roughness added to the ordinary respiratory murmur. This is the *submucous rhonchus*. It may result from slight degrees of bronchitis, and owes its importance only to its

being permanently present when such slight inflammation is constantly kept up by the irritation of adjacent tubercles in an incipient state.

When there is more liquid, not viscid, in the smallest tubes and terminal cells, the rhonchus has a still more crepitating character, and resembles that heard on applying the ear near the surface of a liquid slightly effervescing, such as bottled cider or champagne. This is the *subcrepitant rhonchus* which is heard in œdema of the lungs, humid bronchitis, and other affections in which liquid and air occupy the extreme tubes, and are forced through each other in the motions of breathing.

But the most perfect and equal crepitation is that of peripneumony, called the *crepitant rhonchus*. It exactly resembles the sound which you can produce by rubbing slowly and firmly between your finger and thumb a lock of hair near your ear. I believe that this sound depends on the forcible passage of air, through a little viscid liquid in the finest tubes narrowed by congestion and deposit around them: but we shall speak more of this when we come to treat of peripneumony.

Of all these different rhonchi we may say what we did of the morbid sounds of respiration, that they may occupy the whole of the respiratory movements, or be confined to part of them. Thus an obstruction which is sufficient at the commencement of inspiration to cause a rhonchus, may be insufficient when the tubes are dilated by the distention of a full breath; and you may have the converse, an obstruction which is total in low degrees of respiration and stops all sound, in forced and extensive efforts, as in coughing, occasions a rhonchus. This suggests to us the propriety of using these different degrees of respiration to test the nature and extent of bronchial obstructions. You may also gather from what has been said that the different stages and degrees of force in respiration may change the note of the different rhonchi, and thus produce a variety of music, which we do really hear in the chests of some catarrhal and asthmatic patients. Laennec used to call this piping sound, *rhonchus canorus*. After the explanations which I have given, you may easily perceive that these several rhonchi may be variously combined, or exist at the same time in different parts of the lung, and give rise to numerous combinations that I need not dwell on. I before observed that the loudness of a sonorous or sibilant rhonchus is no proof of the severity of the disease, nor is the fact of its being audible over the whole chest, unless the respiratory murmur be at the same time absent, or very feeble in parts. But the presence of the bubbling or crepitant rhonchi does imply mischief proportioned to its extent; and if they are heard over a large space, and accompanying the whole act of respiration, diminishing or destroying the natural respiratory murmur, they denote disease of a very serious character, because, as your hearing informs you, there is an obstructing liquid in the tubes, where there ought to be only air.

So much for the sounds produced by the passage of air.

We have now another class of sounds to examine—the sounds of the voice as transmitted through the chest. We have found that sounds *produced* by air passing in the lungs are transmitted to the ear on the surface of the chest; and why should not the sounds of the voice, which are strongly *communicated* to the same air, be transmitted in like manner, modified by the size of the tubes, and the nature of the substance through which they pass? So in truth they are, and if carefully attended to, they may thus become signs of the condition of the organs that transmit them.

If you apply your ear to the throat, or upper part of the sternum, of a person whilst he is speaking, you will hear the voice so loud that you might fancy he was speaking into your ear, only the articulation is not as distinct. The reason of this is plain enough. The sound of the voice, although originating in the vibration of the glottis, is propagated to the air above and below it; that below being pent up is not heard without bringing the ear into contact with the parts where the tubes run, and it there resounds with all its force. This is called *tracheophony*, or the natural tracheal voice.

But when the trachea divides and subdivides, there is not only a splitting of the vocal sound into smaller pipes, and a consequent diffusion of it, and a reduction of its volume, but at this division the tubes plunge into the spongy tissue of the lung, which, as we have before found, is a bad conductor, and tends to stop the sound. Hence, over the chief bronchial ramifications, as on each side of the upper part of the sternum, at and between the scapulæ and in the axillæ, you still hear the voice, but it is less in a body, more diffused and more distant, than in the other parts, and the articulation is still less distinct. This is natural *bronchophony*, or bronchial resonance.

What becomes of the voice in other parts of the chest? Why, as it gets into the finer tubes with their more flaccid coats and minute cells, its vibrations are either choked and destroyed, or in some parts may be transmitted across the tissue to the parietes in merely an obscure diffused fremitus. This I call the *pectoral fremitus*. It may be also felt by the hand applied on the chest.

Now before we touch on the modifications of these sounds by disease, I must say a few words on some natural varieties and their physical causes.

Natural *bronchophony*, or the vocal resonance in the bronchial tubes, is most distinct in thin persons with a high or treble voice, as in females and children. Shrill or treble notes penetrate further into the small tubes, because their vibrations are less excursive and need less room than those of a deeper tone. You remember how we noticed this difference in the cords of the guitar. The motions of the treble cords were so short and quick that you could scarcely see them, whilst those of the bass were long and comparatively

slow. Now what will be the effect in a person with a bass voice? Why, the voice will scarcely go into the subdivisions of the tubes, and there will be little or no bronchophony; but if the voice be strong it will not be entirely lost, for it will pass *across* the whole spongy tissue, and throw it all more or less into a diffused vibration, which will be heard and felt in many parts of the chest, in the character of pectoral *fremitus*. We find, then, that treble tones of the voice give more of bronchophony, and bass ones more of the pectoral *fremitus*; and the same occurs with the morbid sounds: if we can get our patients sometimes to change their tone of voice, we may thereby more effectually test the condition of their pectoral organs.

Now, as with the corresponding varieties of respiratory sound, so with these sounds of the voice, they become signs of disease when they are heard out of their proper places. But you will say, as there is some natural variety in different individuals, how shall we know what are their proper places? The diagrams which you see here, and in my work on Diagnosis, before mentioned, and your own anatomical knowledge, will guide you in a general way on this point, by pointing out in what regions the different sized tubes lie; but there is another standard more applicable to individuals, and that is, comparison between the two sides of the chest. As there is an approach to symmetry in the structure on the two sides, so there is in health a general correspondence between their sounds; and as disease scarcely ever affects both sides at the same time, or in the same degree, it will make the phenomena of one side to differ from those of the other. For example, if under one clavicle you hear the voice resound loudly, whilst it is scarcely heard under the other, you may be sure that there is some physical difference between the two sides, which does not exist naturally; or if you hear below the third rib in front of the tubular or bronchial voice, which is generally confined to the immediate neighbourhood of the large bronchi, you may also infer that there is an altered condition of the parts. And now let us see what alterations will change the natural disposition of the sounds.

Increased density of the pulmonary tissue by solid or liquid effusion, or even great sanguineous congestion in it, will improve its conducting power, and will enable it to transmit from the bronchial tubes within, the vocal sounds which they receive from the trachea. This is *morbid bronchophony*, and it is usually accompanied with bronchial respiration. Well, then, if you hear the voice resounding in a part of the chest where it is not usually heard, you may suspect the lung there to be somehow increased in density; but you cannot be sure until you shall have tested this by other means, for there is another change which may have a similar effect. If, instead of the sound being better conducted from within, it is increased in strength and extent by an enlargement of the bronchial tubes, it may then be heard in situations where it does not naturally reach the walls of the chest. In both cases it

may more or less resemble the natural bronchophony heard near the top of the sternum and between the scapulæ; but it often presents remarkable modifications. Thus, when transmitted from the middle-sized bronchi, it comes rather as *diminutived bits of voice* than as articulate words; and for reasons before mentioned low tones are not transmitted; so that if the patient varies his cadence, some words are heard and others not. When arising from dilated air-tubes, or when transmitted from the larger tubes, the resonance is more noisy and continued, varying less with the tone of the voice. If the air-cells over the resonant tubes are still open, the sound will be diminished when they are dilated by a full inspiration, because they then tend to intercept it more. The loudest bronchophony is caused where the middle or upper parts of the lung are pressed against some part of the walls of the chest by a liquid effusion in the pleura, which cannot displace the lung from that part, because it is bound to it by old adhesions.

But what modifies the transmitted voice in the most remarkable manner is a thin layer of liquid between the lung and the walls of the chest. The liquid is thrown by the vocal resonance of the lung into a state of irregular vibration, which causes it to transmit the voice in a broken tremulous manner, so that it sounds to the ear outside like the bleating of the goat. Hence Laennec called it *ægophony*. It may be produced simply by liquid in the pleural sac, without disease of the lung; for the compression of the pulmonary tissue caused by the liquid is enough to enable the lung to transmit the voice from the bronchial tubes within it. When the lung is consolidated also by disease, the vocal resonance is stronger, and there is a loud bronchophony mixed with the bleating voice, constituting a kind of buzzing voice, which Laennec compared to that of *Punch*.

But there is another kind, which may be called the perfection of vocal resonance, in the chest. When a cavity is formed in the lung by the emptying of a vomica or abscess through the air-tubes, the voice will pass from these tubes into it; and if the communication is free, the voice may, by the ear applied outside, be heard in the cavity as loud and distinct as it is in the trachea. This is *pectoriloquy*—not only *voice*, but *speaking*, in the chest. When the cavity is near the surface of moderate size, and opens freely into a large air-tube, the phenomenon is most perfect, and then sounds exactly as if the patient spoke into one's ear; this is limited to the spot where the cavity lies, which is thus, as it were, a little *island* of voice, and when so it is a sure sign of a cavity. The sound of bronchophony is often louder, but then it is more diffused, and there is less distinctness in the words. But I shall not now dwell on these distinctions, because we shall speak of them when we come to treat of the diseases which cause these phenomena.

When the cavity is large, and the opening into it small, the voice may not fully get into it, but there may be a hollow or tinkling reverberation in it, like that in a phial, which is one of the modified echoes from repeated reflection that I told you of in my lecture on

sound. This is called *amphoric resonance*, or *metallic tinkling*, according to the character of the sound. It may be produced in the cavity left by a large vomica or abscess, or by several of these running together ; but its more common seat is the sac of the pleura, into which air has entered through a fistulous opening from the lung. Now, this being the resonant or echoing cavity, you can easily perceive that not the voice only, but the breathing and cough also, especially if they be accompanied by a bubbling through the fistula, will have more or less of this tinkling or bottle sound.

Besides these various positive phenomena of the voice, the absence of the vocal fremitus is sometimes a valuable sign. I have said this pectoral fremitus can be felt as well as heard ; and if you apply your hands, one on each side of a healthy chest, you will feel the vibrations on both sides alike. Liquid in the pleura will generally, more or less, destroy this fremitus, and the difference which it produces is often a very valuable sign of the presence of liquid. Consolidation of the lung, again, will increase the vibrations, or make them stronger over the bronchial tubes. In cases where one side is quite dull on percussion, you may often thus easily distinguish whether the dullness is caused by consolidated lung, or liquid in the pleura—a point of considerable importance in practice.

Besides these sounds produced by the air and the voice, you may sometimes have a sound produced by the motions of the lungs against the ribs. You know that the lungs, although they nearly follow the motions of the chest, do not move quite with it, especially in the lower parts, where the descent of the diaphragm draws the lungs downwards whilst the ribs are rising. But in the natural condition, the surfaces of the pulmonary and costal pleura are so smooth, and so well lubricated with serum, that although there is motion, there is not resistance enough to that motion to cause sound. But suppose these surfaces to become uneven by the deposit of rough matter on them, or by an irregular distension of the tissue by solids or air under them, you may then have a *rubbing sound* with the motions of respiration ; and this does really occur in pleurisy and emphysema of the lung. This rubbing sound is often the more evident in these cases, because the same disease, by preventing the proper expansion of the lung, causes less harmony than usual between its motions and those of the chest.

Now, gentlemen, I have described and explained to you most of the acoustic phenomena of the chest connected with respiration. Those of the heart will be considered in the latter part of this course. Perhaps you may have thought me somewhat minute ; I do not expect you to remember the details of what I have said ; but if your understanding has accompanied me as I proceeded, you must have become pretty familiar with the *principles*, of which the various phenomena are illustrations. I would rather not encumber your minds with too many details, but I do wish to give you a sound knowledge of the laws under which they occur ; for from these, and a good knowledge of pathology, you may understand and deduce, not

only the phenomena hitherto observed, but new forms and combinations of signs which observation and practice may bring before you. In our description of diseases we shall meet with these phenomena again; and from the principles which we have now developed, we shall be prepared for them wherever they may occur. In the mean time, I will leave for your inspection these tabular views of the acoustic phenomena of the chest, and the names by which they have been distinguished. They should be studied in conjunction with others which you will find at the end of my treatise on the Signs and Pathology of Diseases of the Chest.

SOUNDS PRODUCED BY THE PASSAGE OF AIR IN RESPIRATION.

SOUND OF RESPIRATION.—*Natural; produced by collision of the air against the sides and angles of the air-tubes.*

Tracheal; heard in the neck and at the top of the sternum.

Bronchial; near the upper parts of the sternum, between the scapulae, &c.

Vesicular; in most other parts of the chest.

Morbid, modified in production or transmission.

Bronchial, or whiffing; transmitted from the bronchi by condensed tissue of the lung.

Cavernous } produced in morbid cavities communicating with the bronchi.
Amphoric }

RHONCHI, produced by increased resistance to the air moving through the lungs.

Dry; Sibillant } produced by viscid mucus in the bronchi, or by swelling of the membranes, or by pressure upon them.
Sonorous }
Dry mucous }

Moist; Mucous } produced by a bubbling passage of air through } liquid in the bronchi.

Submucous } a liquid in the finer bronchi.

Subcrepitant } liquid in the smallest bronchi.

Crepitant } viscid liquid in compressed smallest bronchi.

Cavernous } liquid in a morbid cavity.

SOUNDS OF THE VOICE TRANSMITTED THROUGH THE CHEST.

NATURAL SOUNDS, *heard in a healthy chest.**Tracheophony, in the neck and at the top of the sternum.**Bronchophony, near top of the sternum, between the scapulæ, in the axillæ, &c.**Pectoral fremitus, in many parts of the chest.*

MORBID SOUNDS, transmitted or produced by a diseased chest.

Bronchophony, transmitted by condensed pulmonary tissue.

Ægophony, the same vibrating through a thin layer of liquid.

Pectoriloquy, resounding in a cavity in the lung.

Tinkling, a changed echo of the voice or cough in a large cavity.

SOUNDS PRODUCED BY THE MOTIONS OF THE LUNGS.

Sounds of friction, when the pleuræ are dry, or rough from deposits.

Emphysematous crackling, by the irregular passage of air between the lobules.



LECTURE VIII.

Physical Examination of the Chest (concluded)—Methods of Auscultation—Directions for Percussion—Pleximeter—Immediate Auscultation—Advantages of Mediate Auscultation—The Stethoscope, its principles and construction—Different kinds of Stethoscope—Directions for using the Stethoscope.

WE are to be occupied to-day, gentlemen, in considering the *methods* of physical examination, or the means which we use to obtain a cognizance of those physical phenomena which we have found to be signs of the condition of the organs within the chest. We have already described the methods of examining the chest by sight, touch, and measurement, and we shall have now only to demonstrate them practically on the living subject. But we have to say something on the methods of hearing, or *auscultation*, and the best means of obtaining the signs which it affords.

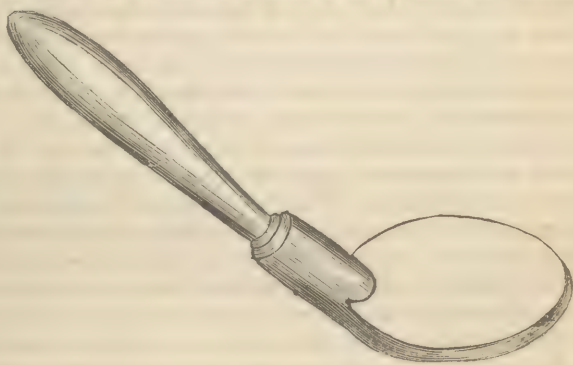
In getting the sounds of percussion you will always hold in mind the principles on which they depend; and when you have to compare those of one part with those of another, you will take care that the parts are struck alike, and that the pleximeter, or fingers, on which you strike, are applied with an equal degree of pressure, and to corresponding parts. Thus, in comparing the two sides in doubtful cases, when you strike on one side on an intercostal space, take care that you strike on the other side on the corresponding

intercostal space, and not on a rib. And when you strike on the clavicle remark well on what part of it you strike ; for the sternal portion of this bone always sounds much clearer than the humeral end. So also in percussing this or any other part, let the direction of your stroke be perpendicular towards the lungs, and not sideways, or you will get the sound modified, not by the lungs, but by the adjacent muscles or other parts towards which the impulse is directed. It is from neglecting this precaution that beginners sometimes get nothing but dull sounds all over the chest. It is not generally necessary to use much force in percussion ; in fact, many of the most valuable results are obtained by gentle mediate percussion in the manner which I before described : but you will remember, from the principles then laid down, that varying the force will give you different results, which in some cases may be usefully consulted. I do not give you any absolute rule whether to percuss with the ends of the fingers, or their palmar surfaces, because both methods are useful in different cases. So also where you want to test the density of a small spot, percussion with a single finger is best ; whilst for trying a surface of greater extent, flat percussion with several answers better. In doubtful cases it may be useful to try both. In percussing the regions of the back and shoulders, seek the bony prominences of the scapulæ and ribs ; for these transmit the impulse to the interior far better than the thick layers of muscles do. But as the scapulæ are moveable bones, it is quite necessary to see that they are in corresponding places on both sides ; and to ensure this, and to increase the tension of the muscles, it is well, in examining these regions, to desire the patient to cross his arms in front, and bow his head forwards. At that part of the chest near the humeral end of the clavicle, a most important region for examination, there is often a falling away of the chest ; and the more tense the pectoral muscles are made, the further they are removed from its walls : here then, instead of making the muscles tense, they must be relaxed, by letting the elbow hang close to the side whilst mediate percussion is practised in this region. There are several other little details with regard to the practice of percussion, that you will soon learn by experience, now that you know the principles.

The best posture of the patient for percussion is erect or sitting ; and in comparing the two sides both before and behind, you should place yourself right opposite to him, and right behind him. When the patient is lying down, the sound is modified by the matter on which he is lying ; if it be a soft feather-bed the sound is more dull, if a mattress or any thing harder, the sound will often be increased because the elasticity of the contents of the chest is increased by the unyielding matter behind it. Never, if you can avoid it, examine a patient in a very empty room, in the corner of a room, or very near a wall, for the sounds are singularly modified by the reverberation of flat surfaces, and the side nearest the wall will consequently give too loud a sound. You may in some measure remedy this by drawing a curtain against the wall, which diminishes the reverbera-

tion : and this is another thing to be attended to, that drapery hanging near the patient will diminish the length of the sound. As most of the effects of percussion are judged by comparison, the chief object of the cautions which I have been giving you is to take care that any of the external causes of modification may not act unequally on the different parts of the chest. If you can, remove the object of your examination beyond their sphere, by percussing him standing in the middle of the room ; but as you cannot always do this, try to equalize as much as possible the modifying circumstances, so as not to affect one part more than another, and you may still get good comparative results.

I do not say much about pleximeters, because I really do not think them generally necessary. If, however, you have a mind to be very well furnished, I recommend you one of this form and size ; it is made of boxwood, and the upper surface of its little plate is covered with soft leather to prevent the clacking sound of the fingers. The handle, rising from its rim at an angle of about 45 degrees, is particularly convenient for holding it firm to the chest, without interfering with the percussing fingers. The chief precaution necessary in using it is, to take care that it be applied flat, and not tilted.



Let us now study the best methods of listening to the audible signs of the motions, the *dynam-acoustics*, of the chest.

All these signs can be heard by the direct application of the ear to the chest ; and this immediate method of auscultation is so easy and simple that it commends itself strongly to us, and is in many cases used with great advantage. The sounds proceeding from the walls of the chest are communicated to the ear, and especially to the air contained in the external meatus, and are thus propagated in the most direct and unmodified manner to the organ of hearing. Immediate auscultation is exclusively practised by some, both at home and abroad ; and as it is much more easily learnt than the mediate method, I have no doubt that it will always have its advocates among those who prefer ease to exactness. You will ask me, what is the use of the stethoscope, if we can hear the signs so well without it ?

I will tell you first some positive objections to immediate auscultation; and on examining the principles of the stethoscope you will find that it has considerable advantages in many cases. To apply your ear, and consequently your nose, face, and so forth, to the chest of a patient who is dirty, or wet with perspiration, you will allow is not pleasant. To apply it to the chest of a patient labouring under an infectious disorder, is neither pleasant nor safe. To apply it to the person of a young female, whether pleasant or not, would certainly not be proper, and perhaps not always safe. Then, you cannot well apply the ear to some parts of the chest, such as the arm-pit, and below the clavicles, or between the scapulæ, in thin persons. Besides this, you will sometimes have disturbing noises from the contact of your hair or your clothes with the patient's chest; and unless your neck be pretty long and flexible, you will find this *easy* method after all more irksome and fatiguing than the mediate method. Still, in a great many instances, it may be used with advantage, especially in examining the regions of the back, and in young children, where the stethoscope might cause alarm, and could not be steadily applied.

We want an instrument, then, to transfer the sounds from the chest to our ear; and what sort of an instrument should this be? It must be a good conductor of sound; and as we found formerly, that the power of bodies to conduct sound will depend on the strength and uniformity of their elasticity, and their capacity to vibrate like the body which communicates the sound, we must have an elastic material, of density resembling that of the sources of the sound within the chest, and of the walls of the chest through which they are transmitted. But the sources of the pectoral sounds vary; some, as the voice and the respiration, or at least the hollower sounds of respiration, are produced in air, whilst in others, such as the sonorous rhonchus, and the sounds of the heart, which we have yet to study, the solids are chiefly concerned: we shall therefore need a varied capacity in our instruments to receive these different sounds. It may perhaps occur to you that as all these sounds have to pass through the solid walls of the chest, their differences must here cease, and their character being identified by being made vibrations of solids, the instrument need only be adapted to these solids. But this view is not quite correct; for the thin and yielding walls of the chest do not considerably modify the character of the vibrations which they receive. As we found by percussion that they derive the character of their vibrations from the nature of the material which lies under them, so when this material originates a sound, the walls vibrating with it do not materially change its nature, or the manner in which it may be communicated to other bodies. It is thus that one parchment head of a drum transmits its vibrations through the air rather than through the solids to the other head, which, in its turn, although a solid, yet freely transmits its motions to the air. In fact, solids, when thin, instead of vibrating only according to their own molecular elasticity, are

carried or moved by the vibrations of the media on each side of them, and their vibrations partake of the character of these media. This is the reason why you can hear voices easily through the thin partition of a room; the sound passes from the air to the partition, and from the partition to the air, and you would not hear any better by substituting for one portion of air a conductor of the same density as the partition itself. I could bring many other illustrations of the same point, but I think I have said enough to show why our instruments for conducting sounds from the chest to the ear should be constructed with regard rather to the origin of those sounds than to the density of the solid walls through which they pass.

To transmit the sounds originating in the solids within the chest, we must have a uniform solid, and the lighter it is the better, provided it be thoroughly rigid. Now nothing answers to this description so well as wood; and in the light kinds of wood with a stiff longitudinal fibre, such as pine wood, cedar, deal, and the like, we find these qualities in perfection. We formerly heard how well a long rod of deal answered as a conductor of the vibrations of a steel tuning-fork. Chladni found by experiment that it is scarcely inferior to steel, glass, and other much denser bodies; whilst it has this advantage over them, that being so much lighter it can be much more readily affected by the vibrations of lighter bodies. Here is a cylinder of deal, about eight inches long and an inch and a half in diameter, adapted to the ear at one end, and through it you may hear most of the pectoral sounds; but those best which originate in the solids, such as the sounds of the heart, of friction, and sonorous rhonchi. You also hear the sounds of respiration and of the voice, but by no means so distinctly as you hear them with the naked ear.

But we want an aerial conductor for these sounds, because they originate in air, and can be best transferred through air. Well, perforate this cylinder with a bore one quarter of an inch in diameter, so as to make a tube of it; if you then apply it you will have a column of air inclosed in the wood, and now you will find that you have the respiration and the voice sounding much more distinctly through it. But as this column of air is in contact with only a small spot of the chest, it can transmit only the sounds produced under or very near that spot, and you have thus an instrument better for exploring small parts of the chest than the ear can be.

We want, however, also, an instrument which will transfer to us the sounds of larger spaces: the sounds of so limited a spot are often too weak to be heard alone; and besides, it would be very tedious to go all over the chest, *dotting* in this way a quarter of an inch at a time. Well, then, let us enlarge our column of air at the base where it is in contact with the chest, by hollowing out the wooden cylinder into a funnel shape, as you see it in this instrument; and by this you will hear the pectoral sounds much more

distinctly than with either the solid or the merely perforated cylinder. Need I explain why? The sounds produced under the whole space covered by the instrument are reflected by the funnel into the central bore, and thus conveyed concentrated to the ear. This also gives the instrument the power of concentrating or magnifying the sounds; and thus you may get them as strong at a distance of several inches, or even a foot or two from the chest, as they are to the ear in close contact with it: nay, in some cases, they are even stronger. But here we have another property required in our instrument, that of reflecting all the aerial sounds which have to be concentrated into the central perforation; and we must think of the shape and material that will do this best. As to the shape, you can easily perceive that a funnel-shaped or long conical cavity, with its apex terminating in the central bore, is the best, because it turns the sound at once in a right direction, without the need of repeated reflections. With regard to the material, we may find bodies that reflect aerial sounds better than wood; such are the metals, glass, porcelain, &c.; and if we wanted particularly to separate this class of sounds from others, these materials would answer best. I have found a stethoscope made with a hollow cone of tin plate or brass answer very well. But these materials are too dense to receive readily the weaker vibrations of the solids of the chest, which are much better transmitted by wood; and wood, although not the best reflector of air-sounds, is quite good enough; for what it does not reflect it transmits: and it matters little whether the sounds all reach the ear by the air, or partly by the air and partly by the wood, so long as they reach it together, which for such short distances they will do. And this leads me to notice another advantage which light rigid wood has over other kinds of material. I told you in my lecture on sound, that the sounding-boards of musical instruments are made of this substance, because its rigidity enables it to receive the finest vibrations of denser bodies, and its lightness renders these vibrations extensive, and capable of impressing a large extent of air, and you saw this illustrated by experiment. Thus it becomes the means of transferring the vibrations from solids to air; and I may now add, the same property also enables it readily to receive vibrations from air. Hence, with a stethoscope of light rigid wood, all kinds of sound are pretty freely transmitted both by the sides of the tube and by the column of air within; and any stray reflections that fall too perpendicularly to be concentrated into the central canal, enter the solid, and are passed on by it; instead of, as in long metallic tubes, being reflected repeatedly from side to side, until they are converted into an echo, which by its tinkle would confuse the original sound.

You see, then, that light rigid wood is the best material for a *stethoscope* or chest-explorer; and the best shape is a perforated cylinder, hollowed at the chest end into a conical cavity, and the other end made flat or slightly concave to fit to the ear. But we

still want sometimes to explore small spots of the chest; and for this purpose we fill up the cavity by a conical perforated plug, which reconverts the instrument into a simply perforated cylinder, which transmits sounds unconcentrated. To make this instrument more portable, the upper part of the cylinder may be reduced to a stem half an inch or less in diameter, leaving only at the top a sufficient width for the ear; or this top may be made separate of a harder wood to slip on and off. Wood is so excellent a conductor of sound, that when once the vibrations are in it, they can be conveyed by a very small body of fibres.

You see, then, that the stethoscope, although a simple instrument, comprehends a good many qualities; the chief of which I will recapitulate.

1. To conduct sound by its solid walls.
2. To conduct and concentrate sound by its closed column of air.
3. To transfer sounds from its column of air to its solid walls, or the converse, when circumstances impede their transmission by one of these ways.
4. To diminish this power of transfer, and contract the field of hearing when small spots are to be explored.

You will find a full description of the stethoscope, illustrated by a plate, in my work on Diseases of the Chest.

If you are not provided with a stethoscope I should recommend you to get one from a turner of the name of Grumbridge, at 42, Poland street. He knows my rules for the construction of these instruments, and executes them well. Here are a great many stethoscopes of all sorts and sizes for your inspection. Do you see this clumsy looking cylinder, made of walnut painted black? Do not despise it, for it was not only made by Laennec, but used by him for several years: and by its means he discovered many of those valuable signs which I rejoice to have to explain to you. With this other mahogany cylinder I began the study of auscultation fifteen years ago; it has done me some service; and I have no small regard for it. The only other instrument that I shall notice, is this flexible one, which is like a common flexible ear-trumpet, only shorter, and with a rather larger tube. As you may suppose, from its variable shape, it does not convey the sounds of the chest so simply or so distinctly as the wooden stethoscope; yet when you become accustomed to it, you can really hear pretty well with it, and its flexibility gives it a great advantage in many cases. It conveys sounds chiefly by the closed column of air which it contains; and the manner in which sound is reflected in curves, as I before explained, enables it to carry the sounds easily through its windings. Yet if you try this experiment carefully, you will find that you always get the sounds more distinctly by keeping the tube as nearly straight as possible. It is particularly useful for separating the sounds of the heart from their accompanying impulse, as I shall explain hereafter.

Before we proceed to our practical demonstrations on the living

model, I must say a few words on the method of using the stethoscope. It is quite necessary that the instrument should be applied in close contact with the chest and the ear; the least tilting uncloses the column of air, and occasions great loss of sound outwardly, as well as a confusing entrance of extraneous noises. To prevent this tilting, it is best to hold the stethoscope by its pectoral end firm on its base, and then to apply the ear flat to the top. If the inequalities of the ribs leave apertures between the chest and the instrument, a fold or two of linen will obliterate these, or the instrument may be used with the stopper in. For the sounds generally it is best to use the concentrating instrument without the stopper; but when it is an object to determine whether a sound is produced in a limited space or over some extent of surface, the circumscribing power of the stopper is wanted. Thus it is often of importance to determine whether a vocal resonance is produced in a small cavity, or merely transmitted by consolidated lung from several bronchial tubes distributed over some extent of surface. The simply perforated cylinder will often do this by showing the size and shape of the limited spot in which the resonance or pectoriloquy of a cavity can be heard in its full strength, while the bronchophonic resonance is transmitted less strongly, and may be traced over some extent of surface, generally in the known direction of these tubes. The stopper is useful also in shutting out the sound of respiration when it is an object to listen to the sound of the heart or arteries, and in many other circumstances which we shall notice as we go through the diseases which produce them.

In conducting your examinations, try as much as possible to avoid fatiguing or annoying your patient. There are cases in which a complete physical examination will do more harm than the information which it may convey can do good; but they are few; and I must leave it to you to hold the balance between too much and too little examination. You will soon in practice find out, too, that your own ease must be attended to in the act of auscultation; for a constrained or painful posture will prevent you from hearing well, and from attending well to what you hear. For this reason you may sometimes hear better with the flexible ear-tube than with the straight stethoscope, although the latter is, for general purposes, the best instrument.

LECTURE IX.

Examination of the Chest through the Vital Properties or Functions of its Organs—Analysis of the general Symptoms of Diseases of the Chest—Dyspnœa; its Nature and Value—Frequent Breathing—Feeling of Oppression—Power of Divers, &c.—Professor Faraday's Mode of enabling a Person to hold his Breath—Table of the Causes of Dyspnœa—Cough; its Nature and Causes—Varieties of Cough, and their Causes:—Catarrhal Cough—Hacking Cough—Irritable and Nervous Cough—Convulsive Cough—Wheezing Cough—Hooping Cough—Hollow Cough—Varieties, from the state of the Bronchial Secretion, &c.

WE have been occupied in the preceding lectures in considering

the physical properties of the chest and its organs, and the manner in which these properties may become signs of the condition of these parts. We have now to examine them through their *vital* properties, which, combined with certain physical and chemical powers, constitute *function*. We have already inquired into the elementary vital properties immediately concerned in the function of respiration—sensibility and contractility,—to which may be added, the power of secretion; and we then found how closely these properties are linked together with the chemistry and mechanism of the organs of respiration, so as to constitute their healthy function. Now any excess, defect, or disorder of any of these properties, will be more or less felt throughout the links of this chain, and hence may arise not only derangement of the function of respiration, or *dyspnœa*, but also new phenomena, proceeding from a loss of due balance of the properties, such as *cough*, *expectoration*, and *pain*; and linked as the vital properties are with those of other organs, there may be added disorders of these, in the form of *disturbance* of the *circulation*, and its sign the *arterial pulse*, *general fever*, *disorder of the secretions of the kidneys, liver, and intestines*, and of the *digestive, nutritive, and sensorial functions*. The phenomena arising from these several disordered properties are what are called the *vital* or *general symptoms* of disease, which we now have to consider in relation to the organs of respiration.

Now it must at once strike you, and I think it will be more apparent as we proceed, that these general symptoms, dependent as they are on such a linking together of many properties, the laws of which are but imperfectly understood, must be far less simple and intelligible than the physical signs; and the variable measure of the vital properties also renders general symptoms far more uncertain than these signs, in their degree, and even in their presence. We cannot with any certainty, as with the physical signs, form a knowledge of the phenomena, and the laws which regulate those phenomena, deduce the condition of the part which produce them, nor from knowing the condition of parts and physical laws, deduce what phenomena the parts ought to develop. For example, the solids of the body have sensibility, which varies not only in different parts, but in the same parts at different times, and this for reasons which we cannot discover; therefore we cannot calculate on it. The contractility of moving parts also varies in a similar manner; and we can by no means gain, from the character of their motions, a criterion of their true condition. Instead, therefore, of pursuing the synthetic as well as the analytic method, which we have done with regard to the *physical* examination of the chest, we shall shortly analyse the chief *general symptoms* of diseases of the chest, and by that examination endeavour to determine their nature and varieties, and their value in teaching us to discover, to measure, and to treat these diseases.

Dyspnœa, difficult or disordered breathing, is the most import-

ant general symptom of disease of the chest, inasmuch as it implies some interruption to the due performance of some part of the great function of the chest—respiration. Dyspnœa may be caused by circumstances affecting any one or more of the several elements concerned in the function of respiration ; viz. the blood in the lungs, the air, the machinery of respiration by which these are brought together, and the nervous system through which the impression which prompts the respiratory act is conveyed from the lungs to the medulla oblongata, and thence to the muscles which move the machinery ; in fact, all the causes which in excess produce asphyxia, in slighter degrees occasion dyspnœa. I will presently show you a table which exemplifies these causes of dyspnœa ; but let us now attend to the character of the symptom itself.

When anything interferes with the sufficient action of the air on the blood, the impression which prompts the acts of breathing not being relieved, causes a quicker and fuller repetition of this act, and if the interference still remain, the breathing will continue to be more or less hurried and forced, until the sensation or impression is reduced to the ordinary standard of almost unconsciousness. Now an individual in whom the breathing is hurried may not be sensible that it is accelerated ; whilst in another who feels the oppression there may be little appearance of shortness of breath. Again, the feeling of dyspnœa must greatly depend on the condition of the sensorium ; for whilst some patients are conscious of the slightest infringement on their respiration, others, particularly in congestive fevers, are brought to the verge of asphyxia without complaining of any oppression. So, too, we are sometimes astonished to find, on opening the bodies of the dead, a whole lung diseased, or one side of the chest full of serum, where the patient had not complained at all of dyspnœa ; while, in other cases, a much smaller lesion of the organs has been attended with the most distressing orthopnœa. It is, however, rather to the sensation of breathlessness than to merely accelerated breathing that the word dyspnœa is generally attached : for translating it as difficult breathing, this expression can be hardly applied when the difficulty is overcome by accelerated movements, of which the patient may not be conscious. But we will not waste our time on words ; but advert to frequency of breathing, as well as the feeling of dyspnœa.

The number of respirations in a healthy adult male at rest generally ranges about twenty in a minute. It is more in children and in females, and it becomes increased in all cases, not merely from affections of the lungs or connected organs, but also from general weakness or depressing causes, which, diminishing the strength of the muscles of respiration, oblige them to make up by the frequency of their contractions what is wanting in their energy. Probably there are some nervous conditions of the system also, in which the breathing becomes accelerated, from what Cullen called *mobility*, a greater readiness to move than power to complete the motions. I

have seen the breathing hurried in some cases of hysteria, without the patient being conscious of it, and without either real weakness or pectoral disease to account for it. These cases are of no consequence in themselves, but should be known, that they may be separated from those of true dyspnœa. In many other cases, especially those, I believe, where the nervous system is affected, the breathing is not accelerated, but suspirious, a sigh or deep breath being taken from time to time; yet the patient is often not conscious of any oppression or unusual effort. This may be called irregular breathing, and there are several other varieties, which we have not time to consider in detail. The rhythm in breathing probably depends entirely on the chain of influences which we before described as concerned in the act, and not on any peculiar periodicity, such as that which seems to reside in the heart; and therefore irregular breathing must depend on a change in one or more of the links in that chain.

The feeling of dyspnœa is one of a very peculiar and distressing character. Even when slight in degree, its permanent oppressive influence is very wearing; and when severe, it causes the most horrible suffering, with such a feeling of impending death, that the most courageous are often unmanned by it. The constrained postures of the patient, the anxious or even desperate expression of his countenance, the painful straining of all the muscles that can in any way, however distantly, assist in the respiratory movements, bespeak the intensity of the feeling, which I have heard several people say is far worse than the most acute pain. It is worthy of remark, however, that this feeling is experienced in its severest degrees only by those in whom the dyspnœa comes on rather suddenly, especially when the sensibility is entire, and the lungs are not diseased, as in obstructions in the trachea or large bronchi, spasm or swelling of the glottis, and spasmodic asthma. In these cases the sensibility is not gradually blunted by the circulation of imperfectly oxygenated blood; nor has the activity of the functions, which require arterial blood, been lowered by previous depressing causes. Opium, belladonna, camphor, and other narcotics, will sometimes relieve the symptoms of dyspnœa, not only by deadening the sensibility, but also by diminishing the activity of those functions and secretions which require oxygenated blood, and therefore a free supply of air. If we could temporarily produce a state approaching to the torpor of hibernating animals, we might diminish the bad effects, as well as the painful feeling of dyspnœa; and I believe that such a state is actually induced in those who are habitually asthmatic, in whom all the functions are brought to a lower standard, and who thus suffer with impunity such an encroachment on the function of respiration as would be fatal to an individual of a common standard.

The feeling of want of breath has been used as a means of attesting the condition of the respiratory organs. A person whose respiration is free and unembarrassed, can hold his breath longer

than one whose lungs are diseased. Some one, I think Dr. Lyons, has proposed to measure the condition of a patient's lungs by the time which he can continue without taking breath after a full inspiration, and to insure accuracy, the patient is desired to count numbers during this time. A healthy person with a good chest can continue counting for forty-five seconds without taking breath, whilst those with diseased lungs often cannot keep on for twenty seconds. The same objection may be made to this test that we made to the measuring of the exhaled air proposed by Abernethy; that it is a test as much for the strength of the muscles of respiration as for the condition of the lungs. Besides, both the feeling of want of breath, and the power of augmenting the respiratory movements, vary considerably in different healthy subjects. It is well known that divers acquire the power of remaining under water for two or three minutes (it has been said more) without taking breath. In diving animals there is a structural provision to enable them to continue some time without air. The chief venous trunks are very tortuous, and admit of dilatation, so that the venous blood can accumulate in them, instead of distending and embarrassing the right cavities of the heart and the lungs. Perhaps some change of this kind may be somewhat produced in divers, by the often repeated practice of holding the breath. Professor Faraday has described another mode by which a person may be enabled to hold his breath for a minute and a half, which is double the time usually practicable. This is by making in succession five or six full and forcible inspirations, which seem to so completely change the air in the lungs, that there is left in them a stock of pure air capable of lasting during that time. The knowledge of this fact may be useful, if ever you should want to hold your breath for a time in going into the suffocating atmosphere of a sewer, a mine, a house on fire, or the like, or in diving.

Dyspnœa is often a symptom demanding great attention in diseases of the lungs; but it must be studied in conjunction with the other general symptoms and the physical signs, for in itself it is most vague and inconclusive. You will perceive this on inspecting this tabular view [see next page] of the causes of dyspnœa, which is founded on the physiology of respiration, as described at the beginning of this course. You will find it worth studying, not only in showing the varied nature and origin of the symptom, but also in contrast with our tables of the physical signs, the causes of which are much less varied, and far more appreciable. But when through the means of the physical and other general symptoms, it has been made out on what cause the dyspnœa depends, then this symptom often becomes a valuable measure of the increase or diminution of the disease, and a useful guide of our practice.

PROXIMATE CAUSES OF DYSPNŒA, OR DIFFICULT BREATHING.

1. BY IMPEDING THE ACCESS OF PURE AIR TO THE LUNGS.

a. *Mechanical.*

Rigidity of parts of the respiratory machine	<i>e. g.</i>	Ossification of cartilages; induration of the pleura; rickety distortions.
Pressure on ditto	<i>e. g.</i>	Tumors or dropsies of the abdomen.
Obstructions of the air-tubes	<i>e. g.</i>	{ Effusions in, swellings of, tumors pressing on, the air-tubes; Spasm of the glottis; spasm of the bronchi.
Compression of the lungs	<i>e. g.</i>	{ Pleurisy, Hydrothorax, Pneumothorax, Aneurism, &c.

Engorgement.

Alterations in the tissue of the lungs	<i>e. g.</i>	{ Effusions Altered structure
		{ { Edema, Hepatisation, Tubercle, &c. Emphysema, Dilated bronchi, Vomica, &c.

b. *Chemical.*

Deficiency of oxygen in the air *e. g.* Mephitic gases; rarified air.

c. *Vital.*

Pain of parts moved in respiration	<i>e. g.</i>	Pleurodyne; pleuritis; peritonitis, &c.
Paralysis of muscles of respiration	<i>e. g.</i>	{ Injuries of the spinal marrow in the neck, &c. Paralysis of the bronchi (?)
Weakness of ditto	<i>e. g.</i>	Excessive prostration, from ataxic fevers, &c.
Spasm of ditto	<i>e. g.</i>	Tetanus; spasmodic asthma, &c.

2. BY THE STATE OF THE BLOOD.

a. *Mechanical.*

Obstruction to the passage of the blood *e. g.* Diseases of the heart and great vessels; tumors pressing on them.

b. *Chemical.*

An excessively venous state	<i>e. g.</i>	Violent exertion; idiopathic dyspnœa (?)
Deficiency of red particles	<i>e. g.</i>	Anæmia; chlorosis.

3. BY THE NERVOUS RELATIONS OF RESPIRATION.

Excessive sensibility of the par vagum	<i>e. g.</i>	Hysteric dyspnœa; cerebral fevers; neuralgia (?)
Defective ditto	<i>e. g.</i>	Coma; narcotism, &c. (breathing slow).

Let us now attend to another symptom, which is even more common in diseases of the chest than dyspnœa is—I mean, the *cough*. The act of coughing consists in one or more abrupt and forcible expirations, accompanied by a contraction of the glottis, trachea, and upper bronchial tubes. The expirations being more complete than usual, especially when there are several of them, are followed by a deep forcible inspiration, the force of which is shown by the loud respiratory murmur, which, by the ear applied to the chest, may be heard to accompany it. The muscles chiefly concerned in the act of coughing are the abdominal muscles and intercostals, as I explained in speaking of the physiology of respiration.

Let us see what is the essential cause of this act: you know that the common cause of cough is phlegm or some other matter irritating the air-passages, and the object or final cause of the cough is to expel or expectorate this matter. Well, I believe we may fairly say that the proximate cause of cough is always some irritation, either direct or by sympathy, of the sentient parts of the air-tube, or of the nerves which render them sentient. Some parts of the bronchial membrane are much more sensitive than others; that lining the glottis and larynx is excessively so, and the least irritation of it is enough to excite coughing. That of the trachea and large bronchi is less, for foreign bodies have been known to lodge in them for some time without causing any coughing, so that some have supposed that they have nothing to do with the production of this symptom; but when the sensibility of these parts is increased by inflammation or nervous excitement, any thing irritating them will also excite coughing. You can easily see why the sensibility of the air-tubes should be greatest at their entrance; it is the *door-keeper*, placed there to exclude, or, by calling other forces to its aid, to expel any thing improper which may intrude. But the other parts of the tubes have also a preserving sensibility, which may bear a little, but is soon roused into activity by continued irritation. We find the parallel of this in the alimentary canal in the natural state. The sensibility that excites the action of vomiting is peculiar to the fauces at one end of the tube; and that which induces the striving of defæcation resides chiefly in the termination of the rectum at the other end; but uncommon degrees of irritation, or an exalted sensibility, will occasion the same actions to be excited by impressions on other parts that are usually insensible: hence arise the vomiting caused by an over-irritated or inflamed stomach or duodenum, and the tenesmus and purging excited by a similar state of the colon. We shall see this more fully on considering the various causes of cough.

As other irritations, cough may be excited either by an unusual irritant acting on the tubes in their natural state, or by the ordinary circumstances, which, although not usually irritating, yet become so by the exalted irritability of the tubes; or, as is the more common case, by a combination of these causes. You have an example of

cough excited simply by an unusual irritant, when a portion of food or of bronchial mucus lodges on the membranes of the glottis ; and an irritant may act by sympathy as well as by direct application, as when you excite coughing by introducing a probe pretty far into your ear. The cause by increased irritability is exemplified in the cough of early bronchitis and nervous asthma, which the mere inhalation of air is sufficient to excite. You have both an unusual irritant, and increased irritability in the secreting stages of bronchitis, and other affections, where an unusual quantity, and sometimes an irritating kind, of mucus is poured out on an over-sensitive membrane. This more complex cause of cough is frequently induced by the continuance of the other causes ; thus, the continued application of an irritant will develop an increased sensibility, and increased sensibility and irritation will be followed by inflammatory excitement and the secretion of matter, the quantity and quality of which add to the irritation. Thus you see how the physiological causes of cough become identified with the pathology of bronchitis, or inflammation of the membrane of the air-tubes ; and in common parlance, you know, a bronchial inflammation is called a *cough*, this being the most prominent symptom. But although this inflammatory condition is often developed by the continuance of causes which produce cough, yet it is not necessarily so, and there may be irritation or increased sensibility, or both, enough to cause cough, and which may yet be short of the degree or the conditions requisite to produce inflammation.

I have said that the irritation which causes cough may not be applied to the bronchial membrane itself, but may be exerted from a part more or less distant. Thus you may have a cough excited by tubercles in the parenchyma of the lungs, by inflammations or irritations of the pleura, peritoneum, stomach, liver, and so forth ; and although we may conjecture that these irritations are conveyed through the nervous branches which connect these several organs and the air-tubes with one common sensitive centre, yet we cannot explain why they should be sometimes conveyed, and at others not ; for although cough does frequently accompany the pathological conditions to which I have just adverted, yet it is very certain that you may often have irritations and inflammations of the stomach, liver, peritoneum, nay, sometimes even of the pleura and pulmonary parenchyma, without any cough whatever. I have heard some one try to explain these discrepancies, by assuming that there must be bronchitis present to produce cough, and that when these several distant irritations do not excite bronchitis, they are unaccompanied by cough ; but this view increases the difficulty instead of diminishing it, for it leaves unexplained the reason why this supposed bronchitis should occur in some cases and not in others ; and bronchitis, although including cough, is more than cough, and needs something more to produce it. We may conjecture about local weaknesses, constitutional peculiarities, and irregular sensibilities, as causes of

these differences, and this is all that we can do towards explaining them, but this is not what an explanation ought to be ; this is referring phenomena not to known general properties, and the laws which govern them, but to individual peculiarities, and undefined influences, the laws of which are not known. These considerations furnish you with another proof of the uncertainty of general symptoms, as means of diagnosis. Still, when cough does occur, and its cause has been made out by the aid of other signs, it deserves attention, not only as a symptom, but as a morbid action of a distressing and hurtful kind, which sometimes may require remedies expressly to relieve it. This illustrates what I have before told you, that general symptoms, although much less constant and instructive than physical signs, with regard to the diagnosis of organic lesions, yet, when positive, often tell us more of those general conditions of the system, which become our guides in the employment of remedies.

Under this impression, I think that we shall do well to examine some of the varieties of cough which present themselves in different cases, and to trace the connexion between their characters, and the variations in elements that constitute them. Of course the study of a symptom in any individual case must be conjoined with a proper survey of its functional or organic cause ; but as we have also (sometimes only) to treat the symptom, it is highly useful to study its varieties, and thus to render it more practically instructive.

The cough may vary according to—

1. The irritant exciting it.
2. The sensibility feeling the irritation.
3. The movements thereby excited, which consist of, *a*, the contraction of the muscles of respiration, and *b*, the contraction of the air-tubes.
4. The condition of the bronchial membrane and its secretion.

Under these heads, we shall meet with varieties of cough with which, I dare say, you are all familiar.

1. When other things are equal, the violence of a cough will be in proportion to the degree of irritation that excites it. For example, a healthy person whilst eating or drinking, incautiously suffers some food to enter the glottis ; the cough thereby excited will be more severe with wine or any thing peppered, than with water or any bland food. So in the early stages of catarrh, although the sensibility of the membrane is increased, yet the thin saline-tasted secretion also acts as an unusual irritant upon it, and keeps up a short teasing, tickling cough, with continued attempts to clear the throat. When the irritation is more moderate, but irremovable, like that occasioned by incipient tubercles in the pulmonary tissue, the cough will generally be of that slight *hacking* kind, with little or no expectoration, that is so well known as one of the first symptoms of pulmonary consumption. The irritant here remaining the same, the circum-

stances which increase this cough are those that augment the sensibility of the lung and air-tubes : such as a quickened state of the circulation from exertion, heated rooms, or during the assimilation of stimulating food.

2. We have already adverted to increased sensibility as being concerned in the cough of recent bronchitis or bronchial irritation. It becomes, however, more developed when the cough has lasted several hours, and instead of being short and tickling only, it comes on in more violent and prolonged fits, which are quite irresistible, and often accompanied by a feeling of soreness. The heightened sensibility of the air-tubes is further manifested by the readiness with which breathing air at all cold, or swallowing any thing at all irritating, will excite cough. We have before remarked how this increased irritability of the inflamed air-tubes is commonly joined with the augmented irritation of their secretion ; but we do sometimes meet with cases in which the increased sensibility is purely nervous, unaccompanied by any secretion ; and the cough is brought on by the slightest cold or irritating matter in the air. Even strong odours will sometimes cause it. These nervous coughs are to be treated chiefly by various remedies which diminish the sensibility of the nervous system, such as narcotics, or sometimes by those which excite stronger impressions in other parts, such as epispastics, and the application of heat.

3. Besides the sensibility of the bronchial membrane, another property connected with the nerves, muscular mobility, may be the source of some varieties of cough. We have no time now to examine the circumstances under which a change of proportionate relation takes place between the action of the motory nerves in general, and the impressions which excite them. It is sufficient for us that the fact is well known, that in certain conditions or states of the system, an ordinary impression will excite inordinate motions ; while in others the motions resulting from similar impressions will be imperfect, and below the natural amount. It is thus also with the motions of muscles concerned in coughing. You may have them excessively mobile, so that the least irritation will set them agoing ; and like a clock without its pendulum, they continue their impetuous motions, until their strength has fairly run out. This is the *convulsive cough*, which we meet with in some hysterical and nervous subjects, and its convulsive character is the more evident from the fact that it sometimes alternates with chorea or convulsive affections of other sets of muscles. The same uncontrollable character is, however, often communicated by a nervous temperament or peculiar nervous affections, to coughs arising from common causes, which thus shake and exhaust the patient in an unusual degree, and require appropriate modifications of treatment to subdue them. *Hooping-cough* in its after-stages is of this kind, and from my experience I should say that the shaking uncontrollable nature of the cough is more characteristic of pertussis than the *hooping*, which is not always present, especially in adults. This leads me to consider on what *hooping* depends, and here again you will

find the use of our physiological divisions which explain some other varieties of cough that are sometimes met with.

In considering the physiology of respiration, we were led to believe that the act of expiration is aided by the contraction of the circular fibres of the air-tubes. In the forcible expirations which constitute ordinary coughing, there is also a simultaneous contraction of the air-tubes, and especially of the aperture of the glottis, through which the air is driven with the greatest force, in order to expel any irritating matter. Now the contraction of these tubes may be excessive, defective, or irregular, and this will occasion other varieties of cough. When their contraction is excessive, being also generally irregular, they give the *wheezing* character to the cough, so remarkable in asthmatic subjects. I do not mean to say that a wheezing cough always depends on contraction of the circular fibres, for other constrictions of the bronchi will also cause it; but if you listen to the chest of a nervous asthmatic, you may often hear, in the forcible expirations of a fit of coughing, sibilant and sonorous rhonchi, which are too transient to be produced by the thickening or secretion of the tubes. Where the irritability of the bronchi is great, their contraction may not, as usual, cease during the act of inspiration; and it is this spasmodic constriction, affecting particularly the upper part of the air-tube during the forcible inspiration which succeeds to coughing, that causes the *hooping* sound. This state of things happens chiefly in the irritable frames of children when affected with convulsive cough, and the violence and repetition of the expiratory efforts of this cough occasion the back draught to be the more forcible, sonorous, and prolonged. If you apply your ears to the chest of a child during a fit of hooping-cough, you will be surprised to find how little sound you can hear there with all these noisy external efforts: in fact, the continued constriction of the bronchial tubes permits very little motion of air into and out of the tissue of the lungs. In the convulsive cough of adults, again, in which there is no hooping, the respiratory murmur of the long inspiration, or back draught, is pretty loud, whilst the succession of coughs here also consists more of external than of internal movement. In all these kinds of cough, antispasmodics will often give more relief than any other class of remedies.

But we may have an opposite condition of the circular fibres of the bronchi, a weakness or deficiency of action, a paralysis, so that they do not contract as usual during the expiratory efforts of coughing. This constitutes the *hollow* or *barking cough* which we sometimes hear in chronic bronchitis, and now and then in febrile and nervous affections. This cough is, as we shall presently find, accompanied with a difficulty of expectoration; hence it is sometimes very distressing, and particularly so when, as it occasionally happens, it is combined with a mobility of the external muscles of respiration, rendering the cough convulsive and paroxysmal. The tearing and exhausting fits of this kind of cough are sometimes quite agonizing; and we may judge from the bloated, congested appearance of the

lips and face, how much these fits impede the respiration and circulation, and how much they may thus tend to increase and perpetuate the diseased condition of the bronchial tubes. In some such cases, I have seen the terebinthinaceous medicines, with external counter-irritation and occasional emetics, give most relief; but the treatment will depend on various circumstances, which I cannot enter into at present.

4. Besides the sensitive and motory apparatus concerned in the act of coughing, we have the secretion of the air-tubes, which may also by its qualities modify the character of the cough. According to whether this secretion is present or not, the cough may be humid or dry; and according to the relation of the qualities of this secretion to the powers of expectoration, the cough may be loose or tight; and these varieties may be combined with the other species of cough, as those may with each other; and thus are produced the endless host of different kinds of cough that we meet with in practice. Without pretending to affirm that you will always be able to classify these by the division that I have now pointed out, I can assure you that I have often found this analysis useful in drawing attention to the predominant changes of vital property, as manifested by this symptom, and in thus distinguishing cases which require different modes of treatment.



LECTURE X.

Examination of the Chest through the Vital Properties or Functions of its Organs.—Analysis of the General Symptoms (continued).—Expectoration; Explanation of the Act; Means of promoting it.—The Matter of Expectoration; its Nature and Value as a Sign.—Morbid Kinds: Mucous; Albuminous; Watery; Compound Varieties.—Signs derived from the Form and Weight of Sputa.—Pain: its Nature and Value as a Sign; its Varieties in the Chest, and their Causes.—Modes of testing the Sensibilities of the Chest.

THE *expectoration* is another symptom of thoracic disease, which must be considered as the result of vital as well as physical properties, and therefore I include it under the head of general diagnosis, although it sometimes approaches in character to a physical sign. Now you know that the word expectoration strictly means the *act of expelling* any thing *from the chest*; but by a figure of speech, it is also applied to the *matter so expelled*. We shall find that both the act and the matter of expectoration may present us signs of the condition of the pectoral organs.

If you consider the structure of the bronchial tree, you may perceive that natural breathing tends to prevent the accumulation of matters in its tubes, in spite of gravitation. The area of the smaller divisions of the bronchi is considerably greater than that of their trunks; and it may be represented as the divided base of a hollow

cone or funnel, which is concentrated in the trunks, and lastly in the windpipe. Now the air, in the more sudden act of expiration, passes with greater rapidity and force as it converges into these trunks, and therefore tends to carry through them any superfluous matter that may be present on the bronchial surface. This will explain how the finer bronchial tubes of the most dependent parts of the lung are, in health, kept clear of any accumulation. Possibly the ciliary motions of the mucous membrane may, as MM. Purkinje and Valentin have surmised, tend to the same effect.

But it is the forcible acts of special expectoration, *hawking*, and coughing, that tend most effectually to clear the air-passages; and they do this by both increasing the force and fulness of the expiratory effort, and at the same time contracting the upper tubes and trachea, so that the air acts with greater force on any superfluous matter in them. The repeated closure of the glottis in coughing increases the expulsive effort by letting out the air in successive sudden jerks, which are more forcible than any continued act of expiration would be. We see this exemplified in cases where the operation of bronchotomy has been performed. The patient often cannot expectorate effectually as long as air can pass out from the artificial opening, and he is in danger of suffocation in consequence; but on closing this during the act of coughing, the force of the air can be directed in the natural way against the accumulated matter. By attention to this particular, suffocation has been averted in more than one instance after this operation. In certain diseased conditions of the larynx, the patient cannot close the glottis; and hence also expectoration may be difficult, and the cough assumes a continuous unguidable character, which we might have added as another variety to those enumerated in the last lecture. This is what M. Trousseau calls a *belching* cough.

There is another element essential to the proper performance of the act of expectoration, the capacity to make such a full inspiration as shall carry the air in beyond the accumulating matter, so that it may on its forcible passage out again carry this matter before it. Hence you see why weakness, which prevents a sufficient inspiratory effort, or obstruction of the terminal and most expansible parts of the air-tubes, which renders this effort ineffectual, may stop the act of expectoration, and by permitting the accumulation of matter in the air-tubes, may speedily conduce to a fatal result. Inability to expectorate is the immediate cause of death in a great many cases of various diseases; in fact, it is a part of the article of death itself; and when you hear the rattle in the throat of the dying, you hear the sign of the accumulating barrier which is shutting out the breath of life. Sometimes, even at this stage, there are sensibilities enough in the system to feel the force of a stimulant which may excite the sinking powers to another struggle; expectoration is once more accomplished, and breath once more renewed; and where there is no irrecoverable alteration of structure, this act of expectoration may in some few instances turn the balance in favour of recovery.

I need not tell you, then, how important it is to study the act of expectoration, and to acquaint ourselves with those means that may excite or promote it. I dare say that every one of you has seen an instance in which a patient has been snatched from the jaws of death by the timely administration of a diffusible stimulus, such as a warm aromatic draught, with carbonate of ammonia, together with such a change of posture and other circumstances as might most favour the expulsion of the matter that was suffocating him. A great deal may often be done in less urgent cases by attention to the posture of the patient. In most instances the act of expectoration is easiest in that posture in which the respiration is most free, which is commonly the semi-erect posture, but I have known some patients expectorate more freely when lying on one side; and I remember a phthisical patient who really appeared to be several times saved from suffocation by alternating his posture from lying down to sitting up in a particular manner, suggested by a knowledge of the condition of the lungs in that case. When this expedient was neglected, the patient was so shaken with frightful fits of fruitless cough, and so oppressed with the accumulating matter, which they could not expel, that speedy suffocation seemed inevitable. In some cases, the act of expectoration may be favoured by another kind of action, in which the expiratory muscles are concerned, that of vomiting; and we shall find hereafter, that some emetics may exert an influence of an important nature on the bronchial tubes, besides this mechanical one.

The *matter of expectoration* frequently furnishes us with very instructive signs. It is the product of diseased action, and in its physical or chemical qualities it may inform us somewhat of the nature of that action, of the condition, and sometimes of the position, of the parts from which it comes. As, however, we have seen that the effort of expectoration is sometimes unsuccessful, so here you will find no matter of expectoration to judge of; and besides this, most children and some adults cannot spit out what they expectorate, but swallow it.

The basis of expectoration generally is the secretion of the mucous membrane of the air-tubes. This is naturally a transparent, colourless, slightly glutinous liquid, like thin mucilage. The chief animal matter which it contains is that called mucus, which seems to be a sort of imperfectly coagulated albumen; and the varieties of sputa presented by disease commonly depend on an unnatural condition or quantity of this animal matter. There is also saline matter, which may vary in quantity, and so may the proportion of water. Unfortunately, we know very little of the chemical differences in the various kinds of sputa; but their physical qualities have been sufficiently studied, to enable us to connect some of them with particular pathological conditions in a way that proves useful in diagnosis and practice.

The difference between mucus and albumen seems to consist in their physical condition, rather than in their chemical constitution.

Mucus is a transparent glutinous matter, not coagulable by heat, as liquid albumen is, and not solid and opaque like coagulated albumen ; but on ultimate analysis it is not found to differ from this principle. When, therefore, we see expectorated matter opaque and solid, or liquid and coagulable by heat, it loses the only distinguishing characters of mucus, and is strictly albuminous.* For this reason, I propose the following general classification of expectorated matter :—

The matter of expectoration may be generally divided into the *mucous*, the *albuminous*, the *watery*, and the *compound* kinds.

1. *Mucous* expectoration is that most like the natural secretion, being transparent, colourless, and more or less viscid. It is the general result of simple acute inflammation of the mucous lining of the air-tubes, in which case it is increased in quantity, and particularly in viscosity ; in fact, the glutinous character of the sputa, and the tenacity with which they stick together, and to the containing vessel, or fall out in a ropy mass, was described by Andral, and I think correctly, to be a mark and, in some degree, a measure of acute bronchitis. In the most intense forms of inflammation, and where the disease occupies the finer tubes, to the glutinous character of the mucus is added a frothiness, arising from the mixture of those air-bubbles in the tubes, which in their breaking cause the mucous and submucous rhonchi. But the most intense bronchitis is that accompanying inflammation of the parenchyma ; here you have the most viscid form of sputum, through which air driven produces the crepitant rhonchus ; and the blood in the distended vessels of the engorged parenchyma communicating a little colouring matter to it, gives it that reddish or rusty tinge which is so characteristic of the sputa of peripneumony. The transparent or semi-transparent condition of these viscid sputa distinguishes them from the albuminous kind, into which, however, they pass in the advanced stages of all the more inflammatory affections of the bronchial membrane. The mucous expectoration has commonly a saltish taste, and with its saline matter is probably connected its irritating quality, so marked in the early stage of bronchitis.

2. The varieties of *albuminous* expectoration are pretty numerous, for I comprehend under this head the opaque kinds of sputa which have no remarkable viscosity, such as the purulent expectoration of chronic bronchitis, the fibrinous or polypous sputa of plastic bronchitis, and the more compound combinations of these with caseous and other matters, which are voided in the advanced stages of pulmonary phthisis. This class of sputa denotes an error of secretion, further than the mucous from the natural standard ; but

* Since these lectures were delivered, an excellent account of the chemical characters of different kinds of expectoration was read at the meeting of the British Association at Liverpool by Mr. Brett.† He has found that the chief character of the sputa of bronchitis, pneumonia, and phthisis, lies in the presence of a variable quantity of albuminous matter, liquid or solid, which does not exist in natural bronchial mucus.

† See *Eclectic Journal of Medicine*, No. 2, Vol. 2, page 40.—Ed.

their production generally announces a decline of inflammation from its most acute form. Probably, the very throwing off of so considerable a mass of animal matter is the means of relieving to a certain extent the inflamed vessels; for we frequently find the purulent or polypous expectoration in intense bronchitis attended by a remarkable diminution in the signs of local and general excitement.* But such an expectoration ceases to be a favourable sign when it *continues*, either with undiminished irritation, or with proofs of general weakness; for then a change is implied, either in the structure, or in the habitual action of the membrane, which, secreting pus instead of mucus, goes beyond the mere removal of a temporary congestion, and proves of itself a cause of irritation and exhaustion. I am not sure that I make myself intelligible, but as we shall revert to this subject in connexion with the pathology of bronchitis, I must dismiss it at present. I dare say you have heard much about the modes of distinguishing pus from mucus in the expectoration. On these formerly the diagnosis of pulmonary phthisis was supposed to depend. We now hold all these tests very cheap, not only because it is well known that pus may be produced without any ulceration or consumption of the lung, but also because these distinctions cannot be complete between matters that pass by insensible gradations into each other.

3. Watery expectoration is that kind in which a liquid of only slightly glutinous quality is coughed up in greater or less abundance. This appears to contain very little animal matter, and to be rather a diluted mucus than to have in it any thing peculiar. It is often covered with a froth, particularly when it is coughed up with much effort. This secretion I regard as the result of irritation, with a relaxed state of the vessels, rather than of inflammation; but it may occur as a consequence of this lesion, as well as of congestion or obstruction to the circulation of the blood in the lungs. It is the expectoration of what is called humid asthma and pituitous catarrh. Some persons of a relaxed habit have it during a common cold, or any form of bronchial inflammation. It sometimes tastes more salt than usual, and in this case it commonly causes a more incessant teasing cough.

4. Under the head *compound* expectoration, I class various combinations of the preceding kinds, which we meet with in almost every form of pectoral disease. They are either products of different parts, in distinct pathological conditions, although coughed up at the same time; or they may in some cases proceed from the same part in an intermediate pathological state, and capable of secreting different kinds of matter. An example of the latter is the opaque or muco-purulent expectoration of the latter stages of bronchitis, in which the opacity and colour of albuminous matter is apparent, whilst it is held together by a mucus of some tenacity. In the *concocted*

* A high authority on thoracic pathology, Dr. Stokes, in his late excellent work, takes a similar view of this subject.

sputa of declining acute bronchitis, the mucus predominates ; whilst the loose albuminous matter is more abundant where the inflammation tends to pass into a chronic state. The sputa of chronic bronchitis, and in fact of most chronic diseases of the lungs and air-tubes, are almost always more or less mixed ; for it generally happens that the different parts of the membranes and tissues are variously affected ; and when, as in the advancing stages of phthisis, there is structural lesion or destruction of parts, there is the greater reason for a more heterogeneous kind of expectoration. In these cases, however, the albuminous kinds mostly predominate, in the form of muco-purulent, purulent, caseous, or tuberculous matter, and coagulable or fibrinous lymph, occasionally tinged or mixed with the colouring matter of the blood ; these constitute the bulk of the expectoration of the consumptive. In catarrhal diseases of a chronic kind, we very commonly see very opposite forms of sputa expectorated together. Thus in a spitting-dish full of thin frothy watery expectoration, you will often find portions of tough and almost solid semi-transparent mucus, as if some parts of the tubes were throwing off the water, and others the animal matter, in a separate form. When we come to the subject of catarrh, we shall find that these opposite products do not imply an equally opposite pathological condition. After hæmoptysis, it is very common to see fibrinous concretions, together with purulent and mucous matter, all more or less tinged with blood. In other affections, it is not uncommon to see the sputa streaked with blood ; and this sign is of less importance when the cough is violent, because it may then merely proceed from a slight abrasion caused by the force of this mechanical action. When, however, there is often blood present, without much force of cough, and especially if there be pus with it, we may suspect the presence of ulceration in some part of the air passages. The colouring matter of the blood, in an altered state, may also be combined with other forms of sputa. Thus, in scorbutic persons affected with humid catarrh, or bronchitis, the expectoration is a thin reddish-brown liquid, like prune juice, or diluted treacle ; and in the last stages of pulmonary disease, the colouring matter, from the final pulmonary congestion which precedes death, is seen in the dirty reddish-brown or greenish tinge of the purilaginous sputa.

You see, then, that the matter of expectoration will often inform us of the pathological condition of the lungs and their tubes ; and its quantity or quality may sometimes suggest proper remedies. In some cases you may learn other things from it. Thus, when in consumption, tubercular matter, with portions of pulmonary tissue, are expectorated, the conclusion is obvious. You will also sometimes see the expectoration present physical signs of the state of the interior by its containing albuminous or compound matter, moulded into the shape of the tubes or cavities from which it comes. The large rounded flocculent muco-purulent sputa of advanced phthisis, are often such as could only accumulate in a cavity ; and the tubular or vermicular albuminous matter which is coughed up in the plastic

kind of bronchitis, sufficiently explains whence it comes, by its being an exact mould or cast of the bronchial tubes, sometimes in an arborescent form, from several of their divisions.

I must not omit to notice a test, which is erroneously used to determine the nature and source of sputa, whether they float or sink in water. Now the floating of a sputum merely depends on the number of air-bubbles retained in it, and although pus alone, or tuberculous matter alone, will not retain these bubbles, yet a small addition of tenacious mucus will enable them to do so. Again, although the sputa formed in ulcerous cavities are less likely to contain air and to float than those formed in the tubes, yet we not unfrequently find the concocted expectoration of acute bronchitis, which is formed exclusively in the large tubes, sink in water; whilst the mixed product of a vomica and the adjoining tubes, which has been churned together with air, floats. This *hydrostatic* test of expectoration is then a very inconclusive one; but it may be useful in sometimes causing a rough separation of the albuminous matters from those of a more viscid mucous kind.

I might occupy you with other details regarding the matter of expectoration; but I have said enough to illustrate how it may prove useful in diagnosis and practice. You will now, I think, perceive how much more valuable its indications may become when conjoined with the physical signs, by which you may often detect the position, and measure the amount of the local disease, of which the matter expectorated is the product. We shall find many exemplifications of this position hereafter.

The only other morbid phenomenon connected specially with the modified vital properties of the organs of respiration is *pain*. Now you know that pain may arise either from an excessive impression on the nerves of sensation, or from an excessive sensibility of these nerves, to which common circumstances of position, motion, &c. then become painful. The latter is the more common cause of pain in internal diseases, but it is not unusual to find them combined, as when a tumour, or effused matter, presses on or stretches parts morbidly sensible. The most common causes of pain are inflammation, and those kinds of vascular excitement that are allied to it; this vascular excitement is generally attended, in the first instance at least, with an exaltation of the nervous function. But the nervous function may be *primarily* excited; and although the increased sensibility thus produced seldom lasts long without more or less stirring up the function of the vessels also, yet we may for a time have pains purely nervous, such as pleurodyne and pectoral neuralgia. Further, as inflammation is not the only cause of pain, so the pain present in inflammation is by no means an index of the extent of the inflammation, nor even of its situation. Most extensive inflammations have been known to occur, not only in the parenchyma of the lungs, but in the bronchial membrane and pleura also, without producing any pain; and it frequently happens in phthisis, that the pains chiefly complained of are low down in the sides,

when the disease is almost entirely in the upper lobes of the lungs. So likewise in bronchitis and pneumonia, the pain is often confined to the sternal, lateral, or scapular regions, whilst the disease occupies other parts.

There are, nevertheless, some general characters with regard to pain, which may render it useful as a symptom of disease of the chest. It is commonly remarked, that the pain of parenchymatous and bronchial inflammations is dull and diffused, whilst that of inflammation involving the serous and fibrous membranes of the pleura and pericardium is of a sharp lancinating character. This is generally but not constantly true; and we may find it explained by the circumstance that the par vagum, which supplies the bronchi and lungs, is by no means so sensitive a nerve as the spinal intercostals, which are distributed on the pleura and pericardium. For this reason, too, there is more apt to be acute pain when the costal pleura, or the coverings of the great vessels, are inflamed, than when the pulmonary pleura is the chief seat of disease. Observe a further distinction in the variations of these various kinds of pain, and you will be confirmed in the opinion that they belong to different orders of sensibility. The dull, heavy, or aching pain of bronchitis, or pneumonia, is generally pretty constant, although it is increased by full inspiration, exertion, or the breathing of cold air; yet even then it gives the feeling of soreness under the sternum, rather than of severe pain. It often resembles the pain of dyspepsia, which is probably seated in a branch of the same nerve, and is also usually referred to the sternum. The degrees of pleuritic pain, on the other hand, are sudden, extreme, and intolerable. If it be not felt in ordinary breathing, a long breath, or a cough, just sufficient to bring the membranes to a requisite degree of tension, causes that sharp stitch of the side—that sudden catch of the breath, that has been considered so characteristic of pleurisy. When it is constant, the patient is obliged to hold his side to diminish its severity, by restraining the motions; and thus placed in opposition to the sensation which prompts the act of respiration, this sharp pain may cause such a voluntary restraint of these acts as to bring the patient to the verge of asphyxia. It is under these circumstances that the breathing becomes partial, as I formerly described to you; and patients whom pain constrains to breathe only with the diaphragm, or with one side, will perform this supplementary respiration so well, that they are completely free from pain, although the inflammation is as acute and the membranes as tender as ever. Sometimes you may detect the latent tenderness by pressing between the ribs of the affected side; but you will be more likely to succeed if you restrain the supplementary respiration by pressing on the abdomen, or on the healthy side, and then desire the patient to cough, or to take a sudden long breath. If there be any exalted sensibility, or tenderness, it is pretty sure to be discovered by this means; and I have several times met with patients who denied

having any uneasiness, or tenderness, yet they winced at the pain developed in this way.

As a general rule (not, however, without some exceptions), we may consider a fixed permanent pain, or a permanent tenderness, which you see depends on the same pathological cause, an indication of inflammation, or congestion, or some analogous condition of the vessels; and when present it deserves attention, not only as an object of treatment on its own account, but also as an index, which, together with the pulse, cough, fever, and other general symptoms, shows the increase and diminution of the complaint, and the effects of remedies, even before these become manifest from the physical signs. Still, if we trust to it alone, it will negatively deceive us in those numerous cases of extensive disease in which it is absent, or scarcely complained of; and it will positively deceive us in those cases in which modified nervous sensibility, a mere neurosis, is the only or the chief disorder.

LECTURE XI.

Examination of the Chest through the vital properties or functions—Analysis of the General Symptoms (concluded)—Symptoms connected with the Circulation—Analysis of the Pulse—Varieties of the Pulse explained—State of the Pulse in Diseases of the Organs of Respiration—Symptoms from the Venous and Capillary Circulation—Symptoms of Fever, or increased heat; Perspiration—Symptoms from the state of the Secretions and other functions—Respective Value of the Physical Signs and General Symptoms.

WE come now to examine shortly the nature and value of the symptoms which diseases of the organs of respiration develop in other functions.

We have noticed, in the physiological part of this course, the close relations which subsist between the organs of respiration and those of circulation; and you will be prepared to expect that disease in the former should disturb the latter, and develop symptoms in the function of circulation. Accordingly we find such symptoms in the state of the *arterial pulse*, and in that of the *superficial capillary* and *venous* parts of the circulation.

You know how much the pulse has for ages been relied on as a guide in the diagnosis and treatment of all diseases; but if you have had much experience, you must know how fallacious it sometimes proves; and if you have had little experience, you will acknowledge that it is very difficult to distinguish the varieties of the pulse from one another. Now I cannot help thinking that some of the fallacies and difficulties connected with the pulse as a sign, arise from our studying it too empirically,—from our not rationally considering those elements on which its varieties depend, and a knowledge of

which would enable us to understand and to foresee the circumstances which are capable of producing these varieties. As the subject of the action of the heart will more fully come under our notice in the latter part of this course, I shall now only detain you with a brief analysis of the nature and varieties of the pulse of arteries.

The arterial pulse, you know, is caused by the jets of blood thrown at certain intervals of time into the arteries by the contractions of the ventricles of the heart. The motion originates exclusively in these contractions, although it may be modified by the blood which is moved, and by the tubes which convey it. Now here you have three elements:—1, the heart; 2, the blood; and 3, the arteries;—and variations in the condition or action of each of these cause varieties in the arterial pulse. Let us consider a few of these variations.

1. Without noticing the modifications in the action of the heart resulting from disease of that organ—a subject to be considered hereafter—it is plain enough, that if the other elements be equal, the strength and frequency, or rhythm, of the contractions of the left ventricle of the heart, will determine the *strength* and *frequency* of the arterial pulse. But the contractions may have another quality—that of abruptness; being rather brisk and short than strong and complete, they communicate to the pulse that character which is called *sharp*. Now what property in the heart gives it this abruptness of contraction? what but an extreme irritability? There is sometimes this irritability in inflammations and fevers; but you may find it also in conditions of mere nervous irritation, of which it is more distinctive. And it is when these co-exist with inflammation or fever, that the pulse presents a sharpness, in addition to other qualities more peculiar to inflammation. In sthenic irritations, or those connected with fulness and tone of the vascular system, which may tend either to acute inflammation or to active hemorrhage or other discharge, the heart's contractions are strong as well as sharp; and so is the pulse. In these cases, although the original irritation were local, it has now reached the centre of the circulation, and, thence, distributed through the whole system, becomes general. But let us see how the other elements modify the pulse.

2. There can be no doubt that the blood in the heart and vessels determines by its *quantity* the character of the arterial pulse; very possibly it does so by its *quality* likewise; but this is not so easy to prove. The fulness and strength of the pulse in the arteries depend materially on the quantity of blood in them; and when the pulse is frequent as well as full, there is the greater proof of plethora, inasmuch as it shows that there is a considerable jet thrown into the arteries at each contraction, notwithstanding that the contractions are so frequently repeated. But you may have a full system of blood-vessels without a large or strong pulse,—as when the heart is acting feebly or faintly; and where its irritability is lowered, such a mode of action may be actually caused by the congestion or distention which for a time oppresses the function until it is roused

into reaction. Under these circumstances, blood-letting will often increase the fulness and strength of the pulse. The opposite condition—a defective quantity of blood—will modify the pulse differently according to the state of the other elements, the action of the heart, and the arterial tubes. When the irritability of the heart is reduced, together with the quantity of blood, the pulse will become softer, weaker, and less frequent. But it frequently happens, especially in nervous temperaments, or where the depletion has been carried to excess, that the diminution of the blood is accompanied with an augmented irritability of the heart, and the pulse becomes not only quicker, but sharper than usual ; and the effect of the abrupt jets into a small bulk of blood contained in imperfectly distended tubes, is to give to the pulse that jerking or bounding character, as if a mere ball of liquid were suddenly shot through the empty tube,—which is so remarkable in the irritation of inanition and chlorosis.

3. But we cannot fully understand the variations of the pulse without attending to the properties of the tubes in which it is felt. If the arteries were tubes of an unyielding or an unvariable character, then the pulse in them would more uniformly represent that of the heart, which would be transmitted through them unmodified. But you know that they are not so : they possess properties of *elasticity* and *tonicity*, which vary according to circumstances, and which modify the pulses from the heart, by changing the size of the tubes, and the yielding or the resisting nature of their walls. Now just think how differently the impulse of a jet of blood must be transmitted by vessels when they are large and yielding, and when they are contracted and tense : in the first case the pulse would be *soft* and *full*, in the latter *hard* and *small*. What are the circumstances which affect the tonicity and elasticity of the arterial coats, and thereby the pulse ? We know these but imperfectly ; further experiments are wanted to elucidate them : but the following are pretty well ascertained, and they should not be forgotten in estimating the signs of the pulse. Cold causes the arteries to contract, and therefore renders the pulse smaller. You know how a cold lotion will often diminish the fulness and throbbing of the arteries of an inflamed part ; and I have seen the same effect of cold more strikingly produced in the large arteries. In the experiments on the sounds of the heart which I carried on in the room below in February 1835, I repeatedly observed, that when the aorta of an ass, recently killed, was plunged into cold water, it contracted, so as not to permit the introduction even of the little finger, and its coats acquired an increased thickness and rigidity ; the pulmonary artery did not contract near so much. The circumstance of temperature must therefore be taken into account in judging of the pulse ; for cold may render the pulse of an artery small and hard, or if severe, small and weak, when the action of the heart and the condition of the system would give it the reverse qualities. Heat, on the other hand, within certain limits, tends to diminish the tonic contraction of the arteries ; so that under its influence they receive

more strongly and fully the pulse from the heart. You know how warmth restored to a limb makes it throb with these expanded pulses.

But there is another circumstance that may modify these actions of heat and cold on the pulse, besides proving by itself a cause of modification—the condition of the capillary circulation. When this is not free, the artery will be more distended, and therefore the pulse harder and stronger than usual; and thus in fevers, where the surface is pale and constricted in the cold stage, and dry and unrelaxed in the hot stage, the pulse often preserves through these changes of temperature a hardness and strength, which would be much more varied were the capillary vessels free and exhaling their usual excretion, and which is actually diminished under the influence of a warm bath or temporary moisture of the skin, although the fever still continue. Again, whatever view we take of the nature of inflammation, we cannot, in the present state of pathological knowledge, doubt that the circulation through the inflamed vessels is to a certain degree obstructed; whilst, either as a consequence of this, or from some co-operating influence, the vessels leading to the part become dilated, and being thus more open than others to the pulse, wave from the heart, they become the seat of that throbbing hard pulse, that has been mistaken for increased action of the vessels themselves. And there are many other variations in the pulse explicable on these principles, but we really have not time to consider them further in detail, as they might occupy two or three lectures.

There is, however, one more cause of variety connected with the arteries, so frequently occurring, that I must mention it:—I mean the difference in the arteries of different individuals. Without any adequate difference in the action of the heart, in the quantity of blood, or in the temperature, you will find a very remarkable variety in the character of the pulse in different healthy individuals; and the same difference extends to the modifications of disease. Some have always a soft large pulse; others a small feeble one; others small and hard; others, again, have habitually a hard strong pulse, which scarcely becomes soft under any circumstances. The first depends on the arteries being large with thin elastic coats. The small feeble pulse may result from their small size and thin coats: this is common in females, and may co-exist with inordinate action of the heart. The hard wiry pulse is connected with small arteries with rigid coats; and the same rigidity or deficiency of elasticity in the coats of arteries of larger size gives that unvarying hardness and strength to the pulse which we so often meet with in old people, and which renders it so uncertain a sign in these cases. You may often, in the radial artery, feel the permanent thickening and hardness of its coats, which thus, like a tube of glass or metal, rigidly transmit the heart's pulses, without tempering them by any yielding or spring. With these peculiarities of pulse there are often connected characters of constitution or temperament, and propensities to disease or health, which are of great importance in guiding us in practice.

I cannot attempt to go into many interesting details which this subject presents; but I hope that the elementary view which I have given may prove something of a key or guide, and that you may now see (for instance) why you are to depend more as a sign of inflammation on a pulse which continues quick and strong, or hard, with permanent heat of the skin, or with perspiration, or through varying circumstances of temperature, than on one which, although very quick, sharp, and jerking, yet without fulness or permanency of character, obviously depends on an irritation more mobile and transient than that of a fixed inflammation. What a capital example of the fallacies of the pulse have we witnessed in many cases of the influenza that has been lately infesting this quarter of the globe! Here you would have a pulse as sharp and frequent as at the commencement of an exanthematous fever; and this with the local symptoms betrayed many practitioners into the supposition that a most serious inflammatory or febrile disease was commencing; but, perhaps, the very next morning you would find the pulse weak, unsteady, and with a moderate degree of frequency; and if, as often happened, an exacerbation took place again at night, the pulse and circulation exhibited the same excessive disturbance, quite disproportionate to the ephemeral character of the fever, and to the degree of the local affection. You would have been tempted by these signs, to use, at one time of the day, active antiphlogistic treatment; at another, stimulating remedies; so rapidly were the functions overbalanced from one extreme to the other, by the excitement and depression which this morbid influence caused in the system.

Besides the general causes which modify the pulse, which we have now briefly considered, there are some specially connected with diseases of the pectoral organs. Those arising from diseases of the apparatus of the circulation will be treated of hereafter. But severe affections of the respiratory organs also sometimes signally modify the pulse, and that in a manner which may tend to confuse its indications. You know how closely the heart is linked with the lungs; by the circulation even more closely than by mere position; for the lungs may be said to lie between the two compartments of the heart; and any considerable obstruction in the lungs will derange the usual relations of these compartments. There is then a distension or over-stimulation of the right side of the heart; while the left, receiving a diminished quantity of blood from the lungs, and that not thoroughly aerated, is less excited than usual, and may give to the arterial pulse a character of weakness and smallness that by no means represents the condition of the whole vascular system, and which often is remarkably contrasted by the action of the right side of the heart, as felt or heard under the sternum. These varieties are produced by any of those affections of the chest which infringe far on the respiratory function. These are more commonly those of the bronchial and parenchymatous kind, which have accordingly been described to be accompanied by a softer and weaker pulse than those affecting the serous

membranes. But you may have a pleurisy also with a small weak pulse, when the effusion or pain is such as to interfere largely with the function of the lungs. Neither are you to suppose that the pulse in severe pneumonia or bronchitis is always weak, even when these affections infringe considerably on the function of the lungs. Even under asphyxiating influences the left ventricle may sometimes become excited, together with the right, and give a sharpness to the pulse, which, combined with the arterial tension of fever, may be readily mistaken for hardness and strength. But this character is seldom permanent; and you will generally find in all diseases, when the function of respiration is much impaired, that the pulse soon loses its body and strength. These considerations suggest the expediency of examining the state of the circulation not only by the arterial pulse, but also by the pulsations of the heart itself, and by the condition of the veins and capillaries.

Under the circumstances just mentioned, when the indications of the arterial pulse are most variable and deceptive, you may often find useful signs in the condition of the *venous* and *capillary* part of the circulation. The distension of the more superficial venous trunks, especially the jugulars, in which a double pulsation often shows also the retropulsive action of an over-distended right ventricle; the fulness of the capillaries of the lips, tongue, throat, cheeks, eyelids, nails, and other parts, at first having a florid and flushed appearance, but afterwards, as the respiration becomes more injured, assuming a purple or livid hue,—are signs of great practical importance, and of a constancy more approaching to that of the physical signs. They do not, however, present themselves in the early and more tractable stages of disease; and they are always less distinct in palid persons with small superficial vessels.

Intimately connected with the state of the circulation is the symptom of *general fever*, or *increased heat*, which attends many diseases of the chest. It depends on increased force and rapidity of the circulation, with diminished perspiration. When the perspiration is restored, the heat always falls. This exhalation of fluid not only lowers the temperature by its physical agency of evaporation, but being in itself a sign of a relaxing of the superficial vessels, it implies an abatement of the vital irritation. In the more transient forms of fever, such as the intermittent and hectic, the profuse perspiration sometimes reduces the animal heat to below the natural standard, just as the circulation is proportionately enfeebled; and you have the same chilling influence illustrated by the cold sweats which succeed to temporary and irregular excitement. There are degrees of vascular irritation in which the increased heat of skin is partial, and determined by the structure of particular parts. Thus in the asthenic excitement of hectic fever, the heat is most felt in the palms of the hands and soles of the feet, because the circulation is not strong enough to drive the perspirable excretion through the thick cuticle of those parts, which become consequently dry and hot. The same thickness of cuticle, on the

other hand, when once imbued with perspiration, often keeps these parts soft and moist, when there is no sensible perspiration on other parts. Not unfrequently the unequal state of the circulation is exhibited in febrile and inflammatory disorders by the heat of the abdomen, back, chest, or head, whilst the extremities are cooler than usual; and occasionally the same locally increased action is manifested by partial sweats, which prevent the increase of heat, and tend to reduce the excitement. I have known a patient with pleurisy perspire profusely only from the affected side, for several days; and nothing is more common, in slight abdominal inflammations, than to find the pungent heat of the belly relieved by a perspiration equally confined to that part. But you know how we practise on the same principle, in applying to irritated or inflamed parts poultices, fomentations, and partial baths, which tend to bring the skin and superficial vessels to the same relaxed and expanded state which they have in a perspiring part.

I think, then, that it will be plain to you that heat of the skin must be an uncertain symptom; for it depends on a condition of the superficial circulation that is by no means constantly associated with disease of the internal organs. When present, it may as much result from a general cause—an idiopathic fever—as from a local inflammation; and cases are not uncommon in which severe, and even fatal, visceral inflammations are attended, through a great part of their course, by free perspiration: nay, the same may be said of some fevers which are called idiopathic. Still the heat and condition of the skin become valuable guides, when taken in conjunction with other signs, inasmuch as they indicate the constitutional disturbance, which is an important part of the disease, and which is sometimes as much to be considered in the treatment as the local disease which has excited it. So, likewise, when the presence of a disease has been established by other signs, the condition of the skin may prove a measure of its increase or diminution more delicate and sooner appreciable than can be found in the physical signs. Thus an increased heat of skin, coming on during a bronchial or pulmonary inflammation, either indicates an increase of that inflammation or the addition of some abdominal or other irritation, which tends to aggravate the condition of the patient. So, too, perspiration breaking out in the hitherto dry and hot skin of a pleuritic patient, occasionally does prove *critical*; whether that word be applied to the excretion as a cause or as a sign of the amendment.

Where there is disorder of the circulation, especially of a febrile kind, you may well expect alteration of the *secretions*, which are so intimately connected with it: hence you find the urine is scanty and high coloured, and the secretions of the liver and intestines variously deranged. As a natural consequence, too, there will be disorder of the digestive and nutritive function: the tongue will be furred, or florid; the appetite will fail; the stomach will cease to digest; thirst will torment; the blood, no longer fed with chyle,

will not duly nourish the textures, nor support the functions ; the strength will fail ; absorption continuing active, if time permit, emaciation will ensue ; and various complications of these disturbances may differently modify the character of diseases of the chest. The sensorial functions, too, may be deranged, either in consequence of the secondary visceral disturbances, or more rarely by a more direct influence of the imperfect respiration on the brain and nervous system. You need only recollect our views of asphyxia, to perceive also how certain states of the nervous system may tend to develop disease of the lungs. Thus insensibility, or coma, causes imperfect respiration, and consequently congestion of the lungs ; and, as we shall hereafter see, a long-continued congestion of the lungs only requires the addition of vascular reaction to convert it into inflammation. Persons rarely recover from an asphyxiated state, without suffering more or less from the injury which it leaves in the vessels of the lungs ; and not a few who have been recovered from suspended animation, have sunk under the pneumonia, or bronchitis, which supervened.

I have been thus rapidly glancing at some of the pathological relations of the organs of respiration to other functions, to give you an opportunity of considering rationally the nature and value of general symptoms. We have said enough of those more nearly related to the chest—dyspnœa, cough, pain, and signs of the circulation, with its concomitant, temperature : although often equivocal, yet when strongly marked, they sometimes assist us in diagnosis. Now it is just the reverse with the symptoms arising out of disorder of the other functions. The altered secretions of the kidneys, the liver and the intestines, cannot inform us of the nature or presence of a disease of the chest ; and still less will gastric derangement or sensorial disturbance. Nay, not only will they not direct, but they tend essentially to blind us to the presence of pectoral disease ; for they set up prominent symptoms of a new character, that may take the attention entirely from the real source of disease, and fix it on the brain, the liver, the stomach or intestines, the affections of which are only secondary, and often trivial. How often do we find a peripneumony, or a bronchitis, disguised by delirium or stupor, or by vomiting, a loaded tongue, or diarrhœa. How often a pleurisy, masked by a jaundiced skin, a tender right hypochondrium, and clay-coloured fæces ; or by a lumbago, or a nephralgia ! How often tubercular consumption, obscured by sundry bilious, dyspeptic, or nervous symptoms ! It will be happy for us, for our credit at least, if not always for the success of our practice, if we detect the enemy through its false colours, ere it triumph, and before the scalpel shall proclaim the delusion of our unwary minds ! The physical signs will enable us to do this, and again I commend them to your most attentive study. We have been occupied for three lectures with the general symptoms, and I trust not unprofitably ; but on taking a rational review of them, in comparison with the physical signs, I think you must come to the conclusion to which my reason and

experience have long brought me, that, as diagnostic means, the general symptoms fall far short of the physical signs.

But do not suppose, that because the general symptoms are often comparatively of little aid in diagnosis, we are to neglect the study of them. They are almost always of great importance in prognosis and practice. The physical signs more surely show how the pectoral organs suffer ; but having discovered this, to the general symptoms we must look for how the system suffers ; and as the system often closely sympathizes with the injured organ, we may, through them, often watch the first turns of the disease before the change in the organ becomes physically appreciable. In the general symptoms we seek for those *critical* phenomena, which, although sometimes deceptive, yet generally announce the tendency of the disease to one or other mode of termination. In them we study the vital forces and properties with which nature works, and the signs of what nature can do ; and in our methods of treatment these become the standards to which we direct, and by which we modify, our remedies. When we treat a patient with peripneumony or catarrh, we do not apply our remedies merely to the local lesions, inflamed vessels, or a discharging membrane ; we study the system at large, we examine other functions through the general symptoms, and we direct our treatment with due reference to indications from all these several sources. You see, then, that the mere stethoscopist is but ill fitted to practise medicine. He may justly boast of his skill in diagnosis : his place of triumph will be the dissecting room, where he can show the lesions that he had detected ; but his practice at the bed-side will be unsuccessful in proportion as local lesions vary in their general relations, and in the conditions of the constitution, or of other functions that may accompany them. The judicious physician will not omit to study the condition of the vital properties, which are exhibited in the general symptoms, as well as the local physical changes which have been already produced ; and whilst he chiefly confides in the physical signs to indicate and measure the present local lesions, he carefully watches in the general symptoms the tendencies of those properties and functions which are capable of increasing or modifying these lesions, and are equally liable to be affected by them. The general symptoms being less intelligible and certain than the physical signs, need more experience to enable us to appreciate them ; but I hope that you now think that even these also may be *rationaly* studied, and may derive a light from a knowledge of physiology and the physical signs, which experience alone could never throw on them.

LECTURE XII.

Diseases of the Air-Tubes—General Pathology of Inflammation of the Mucous Membrane—Bronchitis—Mild Bronchitis; Signs and Course—Treatment of “a Cold” by Palliatives—by Opium—by the “Dry Method”—Severe Bronchitis; its Signs and Course; Treatment—Asthenic Bronchitis—Bronchitis of Infants.

HAVING studied those physical and vital properties of the organs of respiration, which, by their healthy and their disordered phenomena, constitute the *general physiology* and the *general pathology* of these organs, we next come to consider the groups or associations of these phenomena as they are presented to us in *particular diseases*, or special pathological conditions. Now you may judge that we have not time to enter into full descriptions of the various diseases of the chest, nor is it the object of these lectures to do so: these matters are to be found elsewhere, especially in the standard systematic works on the Practice of Medicine, which have appeared within the last few years. Our notice of diseases will be mere outlines with regard to their general symptoms; and it is only where they can derive light from the newer views of pathology and diagnosis, and from the application of these to practice, that we shall be induced to enter into any details; and here I hope that you will find the principles which we have been already considering continually useful as a key and a guide, both in study and in practice.

Following the same course which we adopted in describing the structure of the organs of respiration, we have first to consider the diseases of the air-tubes; and as the most important class of these we shall commence with inflammations.

Bronchitis is essentially an inflammation of the mucous membrane of the bronchi, and it derives its chief characters from the structure and functions of that membrane. Now you know that mucous membranes are less simple in their structure than serous, and the effect of inflammation on them is consequently different; but, as we shall see by and by, the difference does not exclude many parallels. There are, an enlargement of the blood-vessels, an interstitial effusion, and consequent swelling; there is an exalted sensibility; at first there is a diminished, but, as the irritation proceeds, an augmented secretion from the membrane. This secretion is at first transparent and liquid, but more or less glutinous or ropy; and this uniformly viscid quality generally increases with the intensity of the inflammation. No sooner, however, does the inflammation begin to abate, than there is a manifest change in the secretion; it becomes partially opaque and clotted, and although still viscid, in parts even more so than before, it is not uniform, and the masses do not run together and coalesce into a glary mass. This marks the transition of the secretion from the mucous, or muco-serous, to the coagulated albuminous form, which may be simply opaque, muco-purulent, purulent,

or less frequently of a more solid albumen ; and with these changes there are indications of a diminution of the intensity of the inflammation, and of the local swelling and irritation which accompany it. When brought to this state of secretion, the vessels either, having relieved themselves, recover their tone, and gradually return to their ordinary size and function, or if the irritation have continued too long, or have been of a very intense character, they are more permanently altered, and continue to secrete more or less of a mucopurulent or purulent matter, the disease thus becoming chronic.

Now if you bear in mind this general course of inflammation of the bronchial membrane, you will have a key to many of the signs and symptoms of bronchitis ; and the character of the disease will in great measure depend on the stage and the extent of this inflammation, and may thus vary from an affection so trifling as not to interfere with the ordinary occupations of the individual, to one of the most serious and dangerous of diseases. Let us quickly go through the history of the more common varieties of bronchitis.

You know that a common cough, catarrh, or "cold in the chest," is a mild form of bronchitis. This commonly begins with a sore throat or a "cold in the head," the inflammation of which seems to travel down to the windpipe and bronchi, there causing pain or tightness under the sternum, and cough ; at first with a feeling of roughness or dryness in the throat, but afterwards with a tickling, and the expectoration of a thin, glary, salt-tasted mucus, which is often profuse in persons of a relaxed habit : this secretion, so far from relieving the cough, obviously aggravates it ; and I have no doubt that it possesses the same irritating quality that the humour of coryza has, the contact of which with the membrane that secretes it, causes such an unpleasant tickling, sneezing, and lachrymation, and which excoriates the external parts over which it runs. This has also a salt taste, and probably these and all other thin mucous secretions owe their irritating quality chiefly to the saline matter in them. Well, it is when this is secreted that the cough becomes most troublesome, and from its violence there is not unfrequently abrasion of parts of the membrane lining the trachea or larynx, and streaks of blood are seen in the sputa ; these are, however, quite distinct, and do not tinge the mass. Patients are apt to be alarmed at this appearance of blood, but if preceded by a violent cough, this symptom need cause no apprehension. Even mild bronchitis, in its acute stage, is generally accompanied with slight fever, and some shortness of breath ; but I need not dwell more on the general symptoms.

It is by the physical signs that we more clearly mark the condition of the bronchial membrane. In the earliest stage, perhaps before the cough or other symptom of pectoral disease, various dry rhonchi, the sonorous and sibilant, with a diminution of the respiratory murmur, announce the narrowing of some of the air-tubes : more rarely, a total absence of sound in a part of the chest implies that the obstruction there is complete ; but the unimpaired sound on

percussion shows that the vesicular structure is free. These obstructions, no doubt, arise chiefly from the swelling of the mucous and sub-mucous tissues; we see the like place in the nasal canals, when they are the seat of the kindred affection, coryza.* The bronchial tubes do not remain long in this dry state; the secretion commencing first, gives a roughness to the other sounds, and then adds to them a sound of bubbling, which is the mucous rhonchus; but this is seldom so loud as the other sounds, and when the disease occupies only the deep-seated tubes, it may scarcely be heard at all. According as the liquid is in the large or the small tubes, the bubbles, and the crackling which they produce, will be coarse and unequal, or finer and more uniform. The usual seat of all these sounds in the milder forms of bronchitis is in the middle parts of the chest, whether in front, behind, or at the sides, where the larger bronchi lie: in the severer forms they extend to other parts. You may suppose from what I said in a former lecture, in explanation of the sonorous and sibilant rhonchi, that the lower tones imply an affection of the larger tubes; but the acute notes do not exclusively indicate an affection of the finer tubes, for they may be produced also in the large tubes where the obstruction is considerable; and when you hear an acute or whistling note prolonged through the whole act of inspiration or expiration, you may be sure that it is not produced in the finer tubes, for the air is not so long passing through them. I believe that the deep sonorous rhonchus is generally seated at the branching off of a large bronchus, and so strong are its vibrations that it is often felt by the hand applied to the exterior, or by the patient, who can point out the spot where it is. These various sounds may accompany either the inspiration, expiration, or both.

The decline of bronchitis is announced by a looser character of the cough, and the change before named in the expectoration to the opaque concocted kind. This is generally first seen in the morning, that being usually the time of day in which most febrile and inflammatory diseases show a tendency to remission. Sometimes, without this opacity, the sputa assume a more consistent form. This renders the cough and expectoration easier, but it is not accompanied with the general improvement so remarkable when the sputa become simply opaque. In either case, the inspissation of the bronchial secretion causes some change in the physical signs. The bubbles are heard to break more rarely, and give more of a whistling or ticking sound, and the sibilant and sonorous rhonchi become remarkable; but they shift and vary with every cough or forcible act of breathing. The same clots of mucus that by their partial obstruction to the air cause this rhonchi, sometimes totally block up one or more of the tubes, and stop the sound of respiration in the part to which the tubes lead. But this stoppage is seldom

* Dr. Stokes has suggested an additional cause in a spasmodic contraction of the circular fibres, now unduly irritable. It is very probable that this may co-operate.

permanent, and a cough or deep inspiration will often open it, or shift it to another part, and the air is then heard to enter with a whistling or clicking noise, where all had been silent before. The sound, on percussion, is still uniformly good; and this circumstance, with the varying respiration and rhonchi, characterizes bronchitis in this stage.

Before I notice the more formidable varieties of bronchitis, I must say a word or two on the treatment of its common mild kind. "A cold" is generally accounted a trivial complaint; and although the sufferer is often more annoyed and incommoded by it than by affections of a graver name, still it is "only a cold," and he gets little pity, and no treatment beyond that of a few domestic remedies, which may do neither good nor harm. Yet those of you who are liable to colds and coughs, will agree with me when I say that this class of complaints, by their frequent occurrence, by their own evils, and by the many aches and disorders which they often bring with them (tooth-ache, ear-ache, deafness, sore throat, weak eyes, rheumatic pains, indigestion, costiveness, &c. &c.), interfere more with the comfort and occupations of very many individuals than all their other illnesses put together. Surely, then, the subject deserves more attention than it usually gains; and if we cannot make people in general think it worth while to take our advice for a common cold, we may just as well profit by it ourselves. It is a common notion that a cold must run its course; and Laennec cites a proverb much to the same effect:—"A cold well nursed, lasts forty days; a cold not nursed at all, lasts six weeks." I hope, for the credit of our art, that this is not generally true; I am quite sure that I have not found it to be so, or I would not trouble you with any further remarks on the subject. The ordinary mode of treating a cold attended with bronchial inflammation is certainly rather palliative than positively curative: but it generally mitigates its severity, and hastens its termination. A brisk purgative, conjoined (if febrile or gastric disturbance be present) with a moderate dose of calomel and James's powder, will generally take off the edge of the disease; whilst a sedative and expectorant mixture will be useful to quiet the cough, and to lead it to its natural termination by expectoration. In the mild acute inflammations of the air-tubes, the promotion of expectoration is a primary object of the treatment; and besides those means which reduce the intensity of the inflammation (such as sudorifics, evacuants, counter-irritants, and local depletion, some or all of which may be beneficial in particular cases), great advantage will be generally derived from the frequent use of a cough mixture. Various combinations may answer for this purpose. Formulæ of ipecacuanha, or tartarized antimony, with hyoscyamus, conium, or hydrocyanic acid, for the onset; and squill, ipecacuanha, opium, and camphor, for the subsequent periods, are what have appeared to me to answer best. But the efficacy of these remedies is decidedly increased by combining them with an alkali. From ten to twenty drops of the liquor potassæ, or an equal number

of grains of carbonate of soda, or, in more asthenic cases, twenty or thirty drops of the *sp. ammoniæ arom.* are sufficient; and I am confident that in the greater number of cases the alkaline remedies quiet the cough, and promote expectoration far better than the oxymels and acid linctus, or lozenges, that are commonly in use, and which, however they may appear at the time to "cut the phlegm," and cleanse the throat, tend to disorder the digestive organs, and often ultimately to increase the cough. To have their full effect, cough medicines should be taken frequently—at least five or six times a day; for besides that their object is to influence continually the secretion of the bronchi through the circulation, they seem to act in some measure directly on the glottis and its neighbourhood; and in the intervals it is useful to have in the mouth a bit of gum arabic, or of liquorice, the solution of which tends, also by continuity, to sheathe these same irritated parts.

I have nothing further to add to what you must already know respecting this mode of treatment; the great objection to which is, that for its success it requires more or less nursing and confinement. To give diaphoretics and diluents, and at the same time to expose the body to transitions of temperature, which are almost unavoidable without confinement, will tend rather to increase a cold than to diminish it; and I am quite sure that I have seen many colds and coughs kept up by this system. Now, many persons cannot afford the time to confine themselves to nurse a cold; and they either let it run its natural course, or make an even worse compromise, by nursing and sweating one part of the day, and exposing and chilling themselves at another. We want, then, a method of treating a cold, more applicable to those who cannot or will not confine themselves, and of this class especially are medical men. Now I know of two methods of this kind; but to be used with any success they must be employed at the onset of the disease, within a day or two from its commencement, and the earlier the better. The "cold," of which I speak, is an inflammation of the membranes lining the air-passages, accompanied by a secretion which, we have observed, is irritating, and tends to keep up the inflammation. Now, although we cannot cut short this inflammation, and stop this secretion by common evacuants or antiphlogistics, yet we may, by other means, either remove or modify it so as to lead to a more speedy mode of termination. One method that sometimes has this effect is, by taking, at bed-time at the earliest stage of the cold, (whether the affection be felt in the nostrils, the throat, the chest, or in the system generally,) a full dose of opium in some form, and following it the next morning by a brisk cathartic. Ten or fifteen grains of Dover's powder, or two grains of opium with two of ipecacuanha, or half an ounce of compound tincture of camphor, are the most eligible forms of opiate; and I think it is safe to add a few grains of calomel, or some milder mercurial, to prevent the restraining effect of the opium on the secretions. When this remedy acts well, the patient sleeps soundly,

generally perspires freely, and awakes the next morning free from his cold, but often with some headache and nausea, the after affect of the opiate. These are generally relieved by a well-seasoned black draught, and no further ailment is felt but some languor, which another night's rest may remove. I do not know who first suggested this *heroic* cure for a cold; but I believe it is pretty generally practised by members of our profession, and it is by no means an unpleasant one, where opiates and strong purgatives do not disagree. But the stomachs of many do not bear this piece of *medicina perturbatrix*; it may disorder the digestive and alvine functions for weeks afterwards. Sometimes, too, the catarrhal irritation may be too strong to be lulled by the opiate, especially if it be in the chest, and in that case this treatment will hardly fail to aggravate it, and, by suppressing expectoration, it might even convert a bronchitis into a pneumonia.

The other method of stopping a cold is by what I call *drying it up*. I first practised this method in my own person, and you shall hear with what success. In early life I was subject to frequent and most violent colds in the head, which, after lasting a week or ten days, generally ended in a cough, which, even when nursed, I could scarcely ever get rid of in less than a fortnight more; and when neglected, has many times harassed me for double that time. Yet I underwent discipline severe enough; did not taste wine, and scarcely meat, for weeks together, and my chest even now bears the marks of the blistering and tartar-emetic counter-irritation which I inflicted on myself. About twelve years ago, on being attacked with one of these colds, I remarked that taking a quantity of tea, or any other liquid, although very comfortable at the time, was invariably followed in the course of an hour by an increased "stuffing in the head," and accompanying flow of scalding irritating humour from the nose and eyes. I thought I would try to prevent these exacerbations at least, by *cutting off the supplies*—by ceasing to drink. For twenty-four hours I did not take a drop of liquid of any kind, and to my agreeable surprise not only did I escape these occasional aggravations of the complaint, but the stuffing and discharge began to show evident signs of abatement, and the handkerchief was in less continual requisition. I persevered for twenty-four hours more, and my cold was gone; there being only now and then a little gelatinous opaque mucus collected in the nostrils and throat, without any stuffing or irritation, just as it takes place at the very end of a cold. What was of still more consequence, no cough followed; the whole catarrhal disease seemed to have been destroyed. This plan of treatment I have been in the habit of using ever since, with a success varying somewhat in degree, but on the whole amounting to this, that I have had no colds which have lasted three days, and never a severe cough, except once, and that was when circumstances prevented a due perseverance in this dry diet. I have recommended this method of cure to a great many friends and patients; and those who have

had the resolution to use it fairly, have generally succeeded in bringing their troublesome colds to a premature end. You may be ready to say, that the remedy is worse than the disease. I would answer, that either you do not really know what a bad cold is, or your appetites hold a little too much of an epicurean sway over you. I do not recommend the plan to those who have only slight or transitory colds; these may not be worth the penalty of any self-denial; but the amount of discomfort entailed by two days' abstinence from liquids is really trifling in comparison with that and the ill-health that a severe cold brings with it; and I have never hitherto seen any bad effects to arise from the practice. As no account of this method of curing colds has been before the public, except a short notice which I gave in the article *Coryza*, in the *Cyclopædia of Practical Medicine*, I will detain you with a few more observations on its mode of action and application.

The great effect of abstinence from liquids is promptly to decrease the mass of the circulating fluids. The natural fluid secretions continue, although in diminished quantity; the urine is still excreted, although its watery part is diminished; and the skin continues to perspire either insensibly, or, under the influence of increased warmth or exercise, in an obvious manner. Not so the morbid secretion from an irritated membrane: the irritation is lessened with the decreasing fulness of the blood-vessels; the morbid flux no longer continues, the scantier circulating fluid being now taxed for the necessary excretions too closely to supply it; and the diseased membrane, no longer irritated by its own secretion, soon returns to a healthy condition. If liquid be freely taken too soon, before the membrane have lost its diseased action, the discharge will return, and the complaint will be as bad as ever. But if, after the discharge has been stopped by twenty-four or thirty-six hours of dry diet, means be taken to keep up the other secretions, as by exercise, with a warm state of the surface, a little liquid may be taken with impunity, the bulk of the circulating fluid being still below the point at which it can supply any demands from the irritation in the diseased membrane.

I think that the physiological principle of the dry method is what I have now stated; and you can easily perceive that there must be limits to its application. We do not yet know how far it may be pushed, or what may be the extent of its influence on various morbid conditions of the vessels and of secreting organs. I know from experience that partial abstinence from liquids is of great use in moderating many kinds of congestion, hemorrhage, and local determinations of blood, and especially in diseased conditions of the mechanism of the circulation; but we have yet to find out how far a total abstinence may be carried to control acute affection of a similar character. It is necessary also to bear in mind, that other diseases may arise from a defect of liquid in the system; that the excreting organs may suffer from the undiluted excrementitious matters that would pass through them; that their function being

disordered, some of this excrementitious matter may be left in the system, from which very pernicious effects would ensue ; that some functions, as that of digestion, would be arrested by the want of a due proportion of water in the secretions that carry it on ; and other evils which it is not necessary to enumerate. I say, there is reason to expect that such consequences might ensue from carrying this system too far ; but I have never seen any such effects from that amount of the plan which is sufficient to cure the severest cold at its onset. I have more than once passed three whole days without tasting a drop of liquid. The only unpleasant effects were, at the end of the second day, slight headache, with some languor, decided diminution of bodily strength, and a small feeble pulse. These were relieved by a night's rest, returned in the course of the next day, but were entirely removed by two cups of tea at the end of the third day : and all that remained of the effects of the discipline was a desire during the next two days to repair the wants of the system by copious libations, the enjoyment of which was far greater than had been the suffering from abstinence. This suffering is, in fact, rather negative than positive : one certainly does not enjoy one's meals fully without drinking with them ; but this is the diminution of a pleasure rather than an infliction of pain ; and when the meal is over, one forgets all about it. But without dwelling on this point, which being one somewhat of taste, is not to be argued on, let me say a few words more on the application of this plan to the cure of a cold.

It is very essential for the success of the *dry treatment* that the cold should be in its early irritative stage, when it generally occupies the nasal and pituitary membrane. If there be any fever, and especially if the state of the bowels require it, an aperient with an antimonial should be given, for this favours that free state of the secretions on which, as we have seen, the efficacy of the dry plan depends. In milder cases, however, this is not necessary. For the same reason it is expedient that the solid food be not of a too rich or heating kind ; for this, undiluted by liquid, might be apt to disagree. Bread, or any consistent farinaceous food, with a little butter, vegetables, white fish, and white or gelatinous meats, light puddings, and dried fruits, will do very well for a dry diet ; and who can call this starving ? In fact, I have sometimes not changed the diet in any particular but in the point of abstinence from liquids ; and with regard to this point, although total abstinence is, of course, the most effectual, I have found lately that a deviation to the amount of taking about a table-spoonful of tea, or milk, with breakfast and the evening meal, and a wine glassful of water on going to bed, does not interfere with the success of the plan, and it certainly adds to one's comfort. But a great advantage of this plan is, that it does not interfere with one's ordinary pursuits ; it needs no nursing or confinement. In fact, if care be taken to clothe enough to prevent the surface from being chilled, exercise in the open air promotes the success of the plan, by promoting the natural secretions. On the

other hand, those who treat their colds by slops and diluents, which act chiefly by increasing the perspiration, will suffer from the least transition of temperature, which will have an increased influence on a perspiring surface. The time necessary to effect a cure by the dry plan will vary in different individuals, according to the present quantity of their circulating fluid, the activity of their secretions, and the intensity of the catarrhal disease ; and also somewhat according to the temperature and hygrometric state of the air, longer time being always required when the air is cold and damp. On the average, forty-eight hours of abstinence will be quite sufficient. I have often known thirty-six hours sufficient ; but some few severe and obstinate cases required three days. The period may be shortened by exercise and warm clothing, or lying warm in bed, or by commencing with a purgative, or by any other dry means of increasing the natural secretions. The catarrhal affection is generally much relieved at the end of the first day, and only is troublesome at times ; but the cure is not complete till all stuffing is gone, and nothing but a consistent mucus, without irritation, is formed in the nasal or bronchial passages. Sometimes this secretion will continue for a few days ; but unless fresh cold be taken, it causes no inconvenience, and soon ceases. In these cases it is generally prudent to take an aperient and diaphoretic on returning to the use of liquids, which it is always best to begin at night, when there is less risk of relapse from fresh exposure. Perhaps you think that I have been taking up too much of your time with this more trivial form of catarrhal disease, and this novel mode of treating it ; but it is because the disease is thought too trivial, and because the treatment is novel, that I have been dwelling longer on the subject than its comparative importance would seem to demand.

The intense or severe form of bronchitis differs from the milder kind only in the greater extent of the bronchial tubes which it occupies. Its pathological nature and local signs are the same, but its general symptoms differ, inasmuch as the system suffers more from the greater extent of the functional mischief. This disease presents itself in two forms, the *sthenic* and the *asthenic*. In *sthenic* bronchitis, inflammatory symptoms are marked from the commencement : these are generally pain, and a feeling of tightness across the sternum ; hard severe cough, with very glutinous expectoration ; much fever ; heat of skin ; the tongue white and red at the edges ; pulse quick and often hard ; the breathing much shortened, often oppressed, and the least exertion occasions coughing. The physical signs are like those of the mild form, but are here heard more extensively throughout the chest. The rhonchi are at first sibilant and sonorous, afterwards mucous and submucous, reaching to the inferior portions of the lungs, with a weakened respiratory murmur, announcing the presence of the inflammatory mucus even in the smaller tubes ; but the clear sound on percussion declares the vesicular structure still free. If relief be not afforded by expectoration, perspiration, or prompt remedial measures, the disease soon

shows a change of character, from the increased dyspnoea and symptoms of partial asphyxia that ensue. Then come on feelings of great depression ; the pulse becomes weak as well as quick ; the sensorium is sometimes disturbed ; the muscular strength is much reduced ; the countenance becomes anxious and pallid, or partially livid, according to the quantity of blood in the system ; the pulmonary congestion becomes evident by a slightly diminished resonance on percussion in the postero-inferior regions of the chest. The continuance of this state, and the imperfect arterialization of the blood, further disturb other functions ; the secretions become more scanty and vitiated ; the tongue is loaded with a brown fur ; the thirst is intense ; and all these disorders concur in reacting on and aggravating the original disease, and in injuring the natural powers. Such is the loss of balance which ensues from the inroad which severe bronchitis makes on the important function of respiration. The share which the injury of this function has in giving character to the constitutional symptoms, is seen in the fact, that very similar effects are met with in persons who have been subjected to an asphyxiating influence. The step from this condition to death is but a short one, and happens too often, especially in cases that have been neglected at the onset.

Now the advantage of the physical signs is to inform us with certainty of the first coming on of an inflammatory affection of this character. When with the febrile state before described, whether the functional disorder be prominent or not, we find extensive rhonchi in every part of the chest, especially if they extend to the inferior parts, and there be little respiratory murmur audible, we should not hesitate to resort at once to such depletory measures—bleeding, cupping, or leeches—as the individual case will admit, and conjoin with these, mercurial and antimonial medicines to act on the secretions. Generally even moderate bleedings give speedy relief, by removing the congested state of the lung ; and in this respect bronchitis differs from pneumonia, in which this congestion is a more fixed part of the disease. It is desirable, however, to produce an impression on the pulse, which will sometimes increase in fulness as the blood flows and relieves the temporary congestion. But large bleedings are not expedient, for the inflammation of a mucous membrane is not removed by them ; it involves a certain structural change, probably interstitial effusion, that can be relieved only by a free secretion from it. Expectoration is a necessary process during the remainder of the disease, and there must be strength saved for this purpose. In many cases cupping or leeches are to be preferred to general blood-letting ; and they may generally be added with advantage. When the edge of the inflammation has been thus taken off, a very large blister, or the free application by friction of a saturated solution of tartarized antimony on the chest, will keep up the relief given. The latter remedy is well adapted to the more sthenic forms of bronchitis ; and to ensure its prompt action, the chest should be first rubbed with a flesh-brush, or a piece of coarse

flannel : it will then generally bring out a small pustulating eruption in the course of a few hours. Of the internal remedies, tartar emetic in large doses, for the strong, and mercury with ipecacuanha and James's powder, for the less robust, are the most effectual. I shall have an opportunity of noticing the antimonial treatment when speaking of pneumonia ; and I will not now enter into common details of the treatment, as you will find these given in the standard works on practical medicine—as in the article *Bronchitis*, in the *Cyclopaedia*, or more fully in Dr. Copland's *Dictionary of Practical Medicine*.* The good effect of the treatment will be apparent in the general symptoms before it is evinced in the physical signs. The breathing becomes less laboured, the countenance improves, the pulse becomes steadier and fuller. On listening, we may find that the air enters more freely into the lungs, but the mucous and other rhonchi are still present, and continue for some time ; and it is only when the improvement is considerable that we can perceive that they diminish, and that the obstructions become less general ; that instead of bubbling over the whole, or a considerable portion, of the chest, the respiratory murmur is heard, still mixed with clicking, whistling, and humming sounds.

The chief difference presented by the *asthenic* or *humid* form of bronchitis, or *peripneumonia notha*, as it was formerly called, is the early appearance of signs of depression, generally attended with gastric derangement, nausea, headache, &c. ; and by the physical signs we learn, from the universal mucous rhonchus, the early presence of a profuse secretion in all the tubes. I have often observed a temporary dulness on percussion produced by this effusion, or by the pulmonary congestion that ensues in consequence of it : nay, I have known it to cause bronchophony and bronchial respiration, which lasted only during the evening exacerbation, and were gone the next morning. The transient character of these signs is enough to show that the increased density of the lung that caused them results merely from an increased quantity of liquid only, this liquid, probably, being the blood congested in the great pulmonary plexus of vessels, as well as the mucus in the bronchial tubes. This secretion is the great cause of the depression and danger ; and its diminution or free expectoration is a chief object of the treatment. Bloodletting is scarcely borne here ; the most available remedies are large blisters of a size to cover the whole chest ; mercurial purgatives, and tartaric emetic in nauseating or larger doses, with expectorants of a somewhat stimulating kind, such as the decoction of senega, and the liquor ammoniæ acetatis, which do not increase the quantity, but facilitate the act of expectoration. Where the depression is greater, and the act of expectoration begins to fail, more stimulating remedies, such as the carbonate of ammonia, must be given, and the preservation of life will often depend on the judgment with which these are, from time to time, administered.

* To these must be added the valuable and comprehensive chapter on *Bronchitis*, in Dr. Stokes' work, published since this lecture was delivered.

Referring you to the works before named for further details, I shall now only add, that a certain degree of abstinence from liquids is highly serviceable here, not only by diminishing the mass of blood, that has to pass through the now choked lungs, but also, as in the milder catarrh, by reducing the quantity of the bronchial secretion. That it has this latter effect I am convinced by repeated trials; and, on the other hand, I have many times seen a marked aggravation of dyspnœa take place in this and in other diseases of the chest after copious draughts of linseed tea, and other mucilaginous liquids, that are popularly taken with a view to *loosen* the phlegm.

In young children, a very dangerous kind of bronchitis sometimes comes on very insidiously. A certain degree of drowsiness or stupor is present, which keeps the little patient from complaining or coughing. A close attention will, however, detect a great frequency in the breathing; and on applying the ear to the chest, the universal rhonchi, sibilant and mucous, at once declare the latent evil. Emetics and mercurial purgatives are the most successful remedies in these cases. The former must not be too frequently used, as they cause considerable determination to the head, and exhaustion; but they are eminently successful in employing the bronchial tubes of their secretion; and I suspect that they do this not only by the action of the external muscles of expiration, but also by exciting the bronchial muscles to contract, as we know that the glottis is most forcibly closed during the act of vomiting. In the milder cases small doses of ipecacuanha may suffice. The utility of the physical signs is thus most evident in these cases; and in no instance of febrile disease in infants or young children should the practitioner omit to examine the state of the chest.

LECTURE XIII.

Diseases of the Air Tubes (continued)—Chronic Bronchitis: Pathology; Course and Signs; Treatment—Croup: Pathology and Signs; Treatment—Whooping-cough; Signs and Treatment. Diseases of Secretion—Pituitous Catarrh: Pathology and Signs; Treatment—Dry Catarrh: Pathology and Signs; Treatment; Alkaline Attenuants—Bronchial Hemorrhage.

CHRONIC bronchitis is not separated by any distinct line from the acute disease: but in many cases it presents very distinguishing characters. The two forms of inflammation pass by insensible gradations into each other, and are often conjoined; for although acute bronchitis frequently exists alone, chronic bronchitis is rarely free from occasional admixture of acute inflammation, and neither is the long duration of the disease always a proof that it is not acute; for I have known several cases of acute bronchitis in which attack

succeeded to attack for many weeks, and never lost the acute character. The expectorated matter gives us some proofs of the state of the membrane from which it comes ; and by the heterogeneous character often presented by that in chronic bronchitis, we may form a notion of the various pathological conditions simultaneously affecting different parts of bronchial tubes. I think that we may say, however, that there is generally some opaque matter in the expectoration of chronic bronchitis, and that the tendency of chronic inflammation is to make the mucous membrane secrete, instead of glairy mucus, more or less of the opaque matter which we have classed generally under the head albuminous Sputa, whether that be muco-purulent, purulent, fibrinous, or caseous ; whether these occur separately, or, as is more usual, variously combined, of various degrees of consistence and colour, and variously mixed with a thinner and more transparent fluid of a mucous or serous quality.

Chronic bronchitis presents itself in many forms and modes of origin. We cannot stay to enumerate these ; but let us see how it arises out of the acute disease. An acute attack of bronchitis has lasted long enough to injure the membrane ; or its effects have not been controlled by treatment ; or stimulating causes have kept up a general or local irritation at the stage in which the inflamed bronchial membrane was relieving itself by an unusual secretion ; in either of these cases, although the sputa have become partially opaque and *concocted*, and the usual mitigation of the fever and other symptoms have accompanied this change, yet the complaint then becomes stationary, with a lower febrile and inflammatory character, but with unsubdued and more paroxysmal cough, often with dyspnœa, soreness and wandering pains in the chest, and more or less general ill health, and continuing disorder and other functions. The sputa become diffuent, and mixed with some masses, concocted and opaque, of a yellowish or greenish colour, often obviously purulent, sometimes of a dirty grey or brown colour, and in some parts still thin and transparent. More rarely, an albuminous matter, moulded by the shape of the bronchial ramifications, is expectorated by a violent and suffocating cough. When the expectoration is purulent, there is usually a great deal of prostration, and some loss of flesh ; occasionally we find evening hectic, night sweats, and other of the common symptoms of tubercular consumption ; but the physical signs are wanting.

The chest, in simple chronic bronchitis, still expands equally, and sounds well on percussion. The respiration and cough are heard with various rhonchi, mucous, sonorous, sibilant and clicking, which are continually shifting and changing. There is no bronchial or cavernous respiration ; no permanent absence of respiration in a part ; no unusual resonance of the voice ; and in spite of the continuance of the copious and puriform expectoration, on listening, day after day, we still find no signs of a cavity, no cavernous rhonchus or pectoriloquy. Under these circumstances, whatever be the general symptoms, we may pretty confidently pronounce that the dis-

case is not tuberculous consumption, but simple chronic bronchitis. It is not, however, always very easy to get this perfect degree of negative evidence, and it requires much experience in auscultation, or repeated examinations, to enable one to report it with confidence. In such, and all doubtful cases, we should take into consideration also the history of the attack, the constitution of the patient, and such of the general symptoms as may serve to throw light on the prevailing tendencies of the system. The more profuse the expectoration, especially if it be very purulent, or otherwise albuminous, the less likely is the case to be one of phthisis, if no physical signs of this disease are found, and it may be the more readily inferred that the sputa only proceed from a diseased membrane. But there are other difficulties to be considered on this point of diagnosis, which we had better postpone till we treat of phthisis.

Chronic bronchitis is often a very obstinate disease. In its milder kinds it constitutes the winter cough of old people, and may continue for many years without much injuring the constitution of the patient ; but in its severer forms, when the expectoration continues to be profuse, or of a purulent nature ; or when the membranes are extensively affected, and dyspnœa is the result ; or when the cough is very harassing, or has a convulsive character, then the health and the strength of the patient may suffer much ; in fact, those of weakly frames often fall victims to it, and this either from the disease itself only, or also from the alterations which it causes in the structures of the air-tubes and lungs. Of these we shall speak hereafter.

The treatment of chronic bronchitis must vary much, according to the nature of the case, as indicated by the cough, expectoration, and state of the circulation. Bloodletting is not often necessary, unless to relieve a temporary exacerbation or congestion, when leeches under the clavicles, or to the top of the sternum, or cupping between the shoulders, will generally suffice. The most generally useful class of remedies are, counter-irritants, conjoined with mild alterative tonics. Friction of the chest with an oily liniment containing various proportions of tartar emetic, tincture of cantharides, the essential oils, ammonia, acetic acid, or a diluted mineral acid, according to the degree of effect desired ; or a succession of mild blisters ; or, in less severe cases, wearing an ample pitch or mercurial plaster, with a small proportion of blistering fly in it, will furnish a choice of means of counter-irritation, applicable to every case. The methods by friction are preferable to the use of plasters, for they promote in some measure the respiratory movements, whereas plasters, unless they are supple and carefully applied, may somewhat restrain the expansion of the chest. To avoid this the patient should be desired to take long deep inspirations when the plaster is first applied ; and if its material be rigid, it should have long cuts in it from middle to margin, corresponding with the intercostal spaces from the sternum to the sides. When we come to speak of the alterations of structure induced by inflammatory affections, you will perceive the importance of promoting in every

way the free and equal expansion of the chest. With the plan of external counter-irritation it is generally expedient to join such internal remedies as may seem best calculated to improve the condition of the diseased membrane and of the functions generally. These will vary surprisingly in different cases, and although all these cases may suffer from the same local disease—chronic bronchitis—yet they may be best relieved by the most opposite remedies. Mild tonics, such as calumba and cascarilla, with nitric acid, sarsaparilla, taraxacum, are very commonly useful to improve the condition of the secretions and other functions; and where the expectoration is profuse, and even purulent, without much vascular excitement, the mineral acids and metallic astringents in some cases, in others myrrh, copaiba, the balsam of Peru, benzoic acid, and the like, prove occasionally useful. Many of these are safe and beneficial only when combined with external counter-irritation; which, like a safety-valve, lets off in another way any undue stimulation which they may cause. I have found that even steel medicines, particularly that most valuable one the iodide of iron, have been borne, and have sometimes proved very salutary in improving the general health and strength. Of late years much has been said for and against the direct application of remedies, particularly the vapour of iodine and chlorine, by inhalation. I have not much experience of this kind of treatment, but in several cases in which I have seen it tried, an unfavourable effect seemed to arise from the effort necessary in using the inhaling apparatus, the tubes being too small, and the patients complaining of the operation fatiguing them. I should suggest, as a substitute for this plan, the diffusion of iodine, or chlorine, combined with watery vapour, either in the apartment of the patient, or, what would be more practicable, in a small room, or closet, cleared for the purpose, in which he could spend from half an hour to an hour twice a day. Iodine, or chlorine, may be readily dispersed in any quantity through the room, by placing a few grains of the former, or a solution of the chloride of soda, or lime, in a saucer floating on hot water. The quantity should be determined by the effect on the patient; always keeping it below that which causes much coughing or acceleration of the pulse.

When all these means fail, a change of air (especially to a warm sea-coast residence, where the patient can use moderate but regular exercise in the open air) often proves beneficial; and to convalescents, repeated changes, such as those obtained in a sea voyage or gentle land travelling, in warm weather, sometimes prove very salutary. Besides the several symptoms of improvement which I need not enumerate, and the recovery of looks and strength, we should watch for the physical signs, in the more perfect expansion of the chest, the diminution of the rhonchi, the equal respiratory murmur, and the restoration of the expectoration to a simple scanty mucus.

We must just notice some specific affections of the air-passages, in which inflammation is a prominent part, although the accompani-

ing phenomena, or effects, differ from those of common inflammation.

Croup, and membraniferous or plastic inflammation of the air-passages, are characterised by the solid albuminous matter which they throw out ; and their dangerous nature arises from the obstruction which this effusion opposes to the passage of the air. There are two very distinct affections of this kind ; one decidedly inflammatory, of a very acute character, and tending rapidly to the formation of a tough fibrinous matter, which resembles the lymph effused by inflamed serous membranes, but does not appear even to become organized : this is the true croup ; a complaint almost exclusively affecting children. The other affection is more of the character of a cynanche, is less acute, and is more tardily accompanied by the effusion of a soft pellicle, like those which form on the tonsils in a kindred affection ; indeed the tonsils are generally simultaneously affected in the same way. This is the *diphtherite* of Bretonneau ; it is a much more chronic affection than croup : it prevails epidemically, is by some considered to be contagious, and affects adults more than children. I do not think that I have ever seen more than two or three instances of this complaint, so that what I have to say will refer chiefly to croup.

This *croupy*, stridulous character of the sound of the air in passing the trachea, particularly in inspiration, which is the most forcible act, the indistinctness of the pulmonary murmur, owing to the little air that enters the chest, which nevertheless sounds well on percussion, and the concave state of the intercostal spaces at each inspiration, showing that there would be room in the chest if the air could get in ; these are distinctive signs of unmixed croup. But it very commonly happens that there is wheezing in the chest also, from the simultaneous affection of the bronchi with either the plastic or common inflammation. The sonorous inspiration of croup is audible through the stethoscope, or ear, applied to the throat or upper part of the chest, before it can be heard by the ear unapplied : we should, therefore, use this mode of examination with children liable to croup, whenever they show any signs of indisposition. Spasmodic croup is a distinct affection ; but there is probably a good deal of spasm accompanying this disease, both in the early stage, where the inflammation must greatly augment the irritability of the muscular fibres of the air-tubes, and in the subsequent period, when the albuminous coating, becoming detached, acts as a foreign body, continually irritating them. The temporary aggravations of the dyspnœa favour this view. It was remarked by Dr. Cheyne that the solid effusion found in the trachea and bronchi after death, is never sufficient nearly to close these tubes ; in fact it is commonly in a hollow or tubular form, with a pretty large opening within ; it has therefore been suggested as probable that a spasm added to the constriction.

I have nothing new to offer you on the subject of the treatment of croup. At its earliest onset, when the croupy breathing is only audible to the ear applied, bleeding and the warm bath, or an emetic,

followed by a brisk mercurial cathartic, may sometimes arrest the disease ; but when the disease has established itself in its well-known form, that is, after the albuminous exudation has taken place, these remedies lose their effect, and are ill borne. Then the chief resource is mercury in large doses, or tartar emetic watched closely in its effects ; and the success of these measures will depend on there being strength enough to battle against the stifling influence of the disease until their operation reaches the system, and causes the absorption or removal of the effused matter. Sometimes violent and almost suffocating fits of coughing are excited by the retained pellicle. An emetic will sometimes assist in its expulsion. In the less inflammatory affection, diphtheritis, the removal of the albuminous concretions is sometimes accelerated, and a healthy action promoted, by touching the fauces and epiglottis with diluted muriatic acid, or a pretty strong solution of nitrate of silver ; and stimulant inhalations have been recommended with the same view. Dr. Cheyne, Mr. Porter, and other of the best authorities, agree that bronchotomy offers little chance of relief in croup, for the disease generally involves the bronchi as well as the trachea.

The peculiarity of *Pertussis*, or *Whooping-cough*, lies in the nervous, or rather the muscular, relations of respiration. The affection is at first distinctly inflammatory, and the cough is not unlike that of common bronchitis ; but it soon assumes a convulsive character, the fits of coughing being very violent, uncontrollable, shaking the whole frame, and often ending with retching. There may be a *whooping* sound in the inspiration during the cough, but not necessarily ; and this, as we have before noticed, depends on an undue irritability of the bronchial and contractile laryngeal muscles, so that they do not relax as usual during the act of taking breath. During this *whoop*, or sonorous back draught, the ear applied to the chest hears little or no natural respiratory murmur ; but, during the intervals of the cough, this murmur may be as distinct as usual. This does not necessarily imply, however, as Laennec supposed, that there is a spasm in the bronchi ; the upper part of the tube being narrowed, is sufficient to weaken much the sound of vesicular respiration, as it is observed in œdema or spasm of the glottis, and where aneurisms or other tumors press on the windpipe.

I have little to say on the treatment of pertussis in addition to what you already know, or may find in modern works. In the first stage it is almost purely inflammatory : a bronchitis, in fact, but generally combined with more headache and general disorder than usual. In the second stage it is inflammatory or congestive, and nervous ; and at last it becomes entirely nervous ; but even in this stage it is often complicated with alterations in the membrane, and even in the structure of the lungs, which the previous inflammation, conjoined with the mechanical violence of long-continued fits of coughing, has produced. Moderate depletions and other antiphlogistics, in the first stage ; counter-irritants, occasional emetics, and sedatives to the muscular mobility, in the second ; and antispasmodics and nervous

tonics in the third, are the chief heads of the treatment. Stimulant and opiate embrocations rubbed over the chest twice a day, are often very useful in allaying the violence of the cough ; and I believe that they effect this partly by diminishing the excitability of the respiratory muscles. Of internal medicines for this purpose, I know of none so effectual as belladonna ; but, to have its due effect, it should be administered in larger doses than those usually recommended. I have often given a quarter of a grain of the extract three times a day, to a child of two years, half a grain to one of four, and a whole grain to one of eight years of age ; and increased these quantities to double and more when they ceased to relieve. These doses generally cause some dilatation of the pupil ; and I conceive that the remedial agency of the drug depends on the same power to diminish irritability in the bronchial and laryngeal muscles, which is here evinced with regard to the iris. In some cases there have been some feeling of heat and dryness in the throat, giddiness and pain over the eyes ; but these symptoms lead to no bad consequences, and soon pass off when the medicine is discontinued. In fact, I have known more than one instance of a large quantity of the extract of belladonna being swallowed in mistake (in one case upwards of a drachm), without any other bad effects than the temporary production of the symptoms described as those of *poisoning* by belladonna, and which entirely passed away in a day or two. Sir Benjamin Brodie has informed me that he has repeatedly given large doses to animals ; and although they manifested for a time many ugly symptoms of nervous disturbance, they all ultimately recovered. I tell you this, that you may not be alarmed should untoward symptoms sometimes manifest themselves during the exhibition of belladonna. They occasionally occur when it is given in very moderate doses, and even during the application of a belladonna plaster externally ; but they do no further harm. In many cases I have found the belladonna to diminish signally the violence and number of the paroxysms of hooping-cough ; but as it loses some of its efficacy by continuance, I have found it best to intermit its use for a few days, and then to resume it again. In the more violent cases we are obliged to resort to opium, which, if more powerful as a narcotic, also may do more harm than belladonna. The preparations of morphia are perhaps the best form of opiate, and they should be combined with ipecacuanha. In the latter stages a change of air is almost a specific ; any kind of change, and although only to the distance of a few miles, will sometimes entirely remove a cough that has baffled all medicines.

In the convulsive coughs of adults, which resemble hooping-cough, I have several times found the pilula aloës et asafœtidæ twice a day, with a galbanum or pitch plaster to the chest, cause most effectual relief. Where the complaint is more obstinate, and the nervous system takes up the habit (as in chorea and other convulsive diseases), the metallic salts, subnitrate of bismuth, nitrate of silver, or ammoniaret of copper, will sometimes succeed in restoring the balance ; and they may generally be aided by the shower

bath, country air, and exercise, and other means which diminish the mobility of the nervous system.

We have seen how remarkably inflammation alters the secretion of the bronchial membrane; but it may also be altered without there being any distinct signs of inflammation. Thus in cases of what in this country is generally called humoral asthma, the attack and its removal are too sudden to be ascribed to inflammation; and after death there are not found any appearances that betoken an inflammatory character; nay, the membranes are often paler and thinner than usual. Although such affections may originate in, or be occasionally complicated with, bronchitis in some of its forms, yet they are not essentially so, and may with more propriety be called diseases of secretion. The most remarkable of these are the *pituitous* and *dry catarrhs* of Laennec.

Pituitous catarrh, bronchorrhœa, or bronchial flux is remarkable for the quantity of thin liquid that is discharged by expectoration. This liquid does not appear to differ materially from the natural secretion, but in being thinner, and it is probably this, diluted with a considerable addition of the most watery part of the blood. This suggests that a laxity of the bronchial vessels may be its immediate pathological cause; and this view is confirmed by the fact that we find it to occur chiefly in those of a relaxed habit of body, who usually drink much liquid, and perspire profusely. I have seen several cases in which a discharge of this kind was plainly vicarious with the exhalation from the skin, sudden changes of temperature, irregularities of diet, disorder of the digestive functions, which depressed the cutaneous circulation, being sufficient to excite it. It is often combined or alternated with a similar discharge or phlegmorrhagy from the nasal membrane, a sort of chronic cold in the head. Sometimes it is connected with organic disease of the heart, which causes congestion in the lungs. It generally comes on without fever, with sneezing, cough, and asthmatic or wheezing breathing. On applying our ear to the chest, we hear all sorts of sonorous and whistling rhonchi, which, as the attack proceeds, become mucous or bubbling; and very little of the natural respiratory murmur is heard. The sound on percussion is generally pretty good; but in severe cases this is also impaired by the profuse quantity of liquid, which from the submucous and subcrepitant rhonchi may be known to extend even to the smaller bronchial tubes. Coming on, as the attack does, suddenly, the dyspnoea is sometimes extreme; but the strength of the respiratory forces being also unimpaired, they generally, by dint of violent coughing, get rid of the fluid as fast as it is secreted, and it comes up clear and frothy, sometimes to the amount of a pint or more.

Although not generally dangerous, this complaint often causes much suffering and discomfort; and if it occur frequently it causes much weakness and exhaustion. This affection is sometimes very obstinate in regard to treatment, especially when it has become established in the habit. The most powerful means are those of

diet and regimen. A well-regulated, nourishing, but not stimulating diet, with a limited quantity of liquid ; a bracing, but not too cold air ; and, above all, regular and pretty active exercise, with clothing always sufficient to protect the surface from transitions of temperature, and to favour an equable moderate perspiration, will often do more than medicine. Of remedial agents, mild tonics, the mineral acids, or some metallic salts, such as the sulphate of zinc, in small doses, and the milder preparations of steel, are sometimes beneficial. In other cases some of those medicines supposed to act especially on the mucous membranes prove serviceable such as ipecacuanha, the balsams of copaiva and Peru. During the paroxysm, as well as in the stage of effusion in humid bronchitis, I have known the ethereal tincture of the *lobelia inflata* very materially relieve the dyspnœa, and shorten its duration. It is, however, a very uncertain medicine, sometimes causing vertigo and nausea in the dose of ten minims, in others giving relief only in the dose of a drachm. There are many other remedies that now and then appear to do good, such as emetics, opium, blisters, &c., but I have no time to enter into further details on the empirical part of the subject.

The other affection which exemplifies the altered secretion of the bronchial membrane without distinct inflammation, is the *dry catarrh* of Laennec. This term, *dry catarrh*, is a very bad one ; and but that the word catarrh is now used almost synonymously with cough, it would be totally inapplicable. Catarrh means a *flowing down* ; and what can be greater nonsense than *dry flowing* ? The symptoms of this affection are those rather of asthma than of bronchitis. They vary according to the extent of the affection. In its slightest degrees, it is presented by those individuals who, every morning on waking, feel their breath rather short until they have coughed up a little tough semi-transparent mucus. In its severer degrees, that is, when more of the bronchial membrane is affected, the shortness of breathing may amount to a regular fit of asthma, accompanied by cough ; and this may last more or less, for hours, and even days, and be at last relieved by the expectoration of the scanty tough expectoration just mentioned. There is little or no fever or sign of inflammation present ; only sometimes a sense of constriction and heat, or rather of stuffing in the chest ; but there is often much gastric disorder ; the tongue slightly furred ; the uvula relaxed ; the tonsils congested ; digestion imperfect ; the liver inactive ; the bowels torpid, or liable to extremes ; the hemorrhoidal veins swelled ; and the urine turbid. Excesses in diet, the sudden removal of cutaneous eruptions, suppressed gout, and sudden checks given to perspiration, or any other free secretion, occasionally excite this affection. These causes operating on systems not much disposed to inflammatory reaction, such as those of a torpid habit of body, destroy the balance of the capillary system, and occasion an undue distension or congestion of certain parts of it. This congestion is accompanied by a disorder of the functions of the part, and in the bronchial membrane, especially by

a derangement of its secretion. The same kind of passive congestion is sometimes more directly occasioned by organic diseases of the heart, particularly those in which there is some obstruction in the left ventricle ; and here we frequently have the symptoms of dry catarrh.

The physical sign of this disease is a more or less complete suspension of the respiratory sound in the part affected, whilst the chest, at that point, still sounds quite well on percussion. This suspension is caused by the tumefaction of the bronchial membrane, which either of itself, or assisted by the scanty thick mucus before named, obstructs the passage of air in ordinary respiration. Sometimes, during coughing, or violent efforts of respiration, a wheezing or sibilant sound announces that the obstruction is not quite complete : and there will generally be some of the tubes which will give these sounds during common breathing. These signs, and the tough scanty expectoration, characterise this affection ; to which I would give the more pathological name *bronchial congestion*. Coming on, as the attack sometimes does, suddenly, continuing for a few hours or longer, and then as suddenly ceasing, it bears none of the characters of inflammation ; but the swelled state of the membrane and its dark colour without other change, which I have seen exhibited after death, imply a full state of the blood-vessels referrible to the class of congestions, which may be produced and endure for an indefinite time, and have not the same tendency to definite terminations that inflammatory injections have. It may doubtless, originate sometimes in inflammatory affections of the same part ; but according to my experience it is more commonly the result of disorders of the digestive or other organs which tend to injure the tone of some, or other part of the capillary system. Thus, these will, in some persons, locate this congestion in the capillaries of the face, harming nothing but their beauty ; in others the encephalic vessels suffer, whence habitual headaches of an obstinate character arise ; in others some part of the alimentary canal is the seat, whence indigestion, hemorrhoids, or some disorder of the alvine function, ensues. So, too, the urinary or the genital systems may become the place of this congestion ; or it may fall on the bronchial membrane, and induce the affection under consideration ; and the local determination of the morbid vascular condition is, in individual cases, fixed on particular parts or organs in consequence of prior weaknesses or tendencies, or other circumstances which we cannot now stop to enumerate.

Occasionally bronchial congestion is conjoined with what might seem to be its opposite, pituitous catarrh ; but according to the view which we have taken of that affection, the pathological causes of the two do not differ very widely, the same circumstances causing a loss of tone in the capillaries, being capable of producing a relaxation of their exhalant properties, or a dilatation of their caliber. Or what is more usually the case, some parts of the membrane are affected with one, and some with the other ; and the result is the expectoration of much thin glairy fluid, with the little pellets of tough mucus

in it. So, too, by a modification in the properties of the congested vessels, they may be excited or relaxed, and relieve themselves by the exhalation of their watery contents; and we accordingly, sometimes, find an attack of catarrh or asthma, at first quite dry, and devoid of any but the tough expectoration, suddenly relieved by a copious discharge of thin frothy fluid. This happens more commonly where the congestion results, mechanically, from disease of the heart.

Depending, as this affection generally does, on constitutional causes, it will require measures which may act on the system, as well as those which may improve the condition of the affected membrane. A due management of the diet, avoiding all acid, rich and irritating articles of food; regulating the secretions by mild aperients and alteratives; and subsequently employing tonics, which, by maintaining the balance, may increase the general tone of the circulation, generally constitute the most important part of the treatment. With regard to the measures addressed to the congested membrane, it is not found that the ordinary remedies for bronchitis are of much avail. Blood-letting produces little impression. Dry cupping, and other means of derivation, may be of more use. Stimulating and bracing applications to the whole surface of the chest, pitch plasters, flying blisters, and stimulating embrocations, are occasionally of temporary advantage.

There are, however, means of increasing the flow of the bronchial secretion, which, as temporary remedies, are of more avail than any hitherto named. We have before noticed the property which alkalies have of determining to the bronchial surface, and we have now to notice in addition their attenuant or dissolving power, which diminishes its tenacity, augments its quantity, and thus facilitates its expulsion. These remedies thus not only remove the obstructing mucus already secreted, but by favouring its looser flow, they tend to unload and reduce the congested membrane, and thus to relieve the dyspnœa that arises from its tumefaction. For the knowledge of these remedies I am indebted to Laennec, who seemed, however, at a loss to account for their efficacy. I am far from wishing to extol chemical medicines in general; but I cannot but think that we may bring chemistry to our aid in the present instance, to explain the action of alkaline attenuants. We know that we can, by the administration of alkaline medicines, render the urine alkaline, and increase the alkaline qualities of the blood. Now there is no solvent of mucus more effectual than alkalies, and it is easy to perceive that an alkaline state of the bronchial secretion can scarcely be compatible with the formation of tough solid mucus. I have found these remedies very effectual, and I am in the habit of giving either the liquor potassæ (℥.xx. to xxx.), carbonate of soda (gr. xv. to xx.), or carbonate of ammonia (gr. iij. to vj.), according to the character of the case, three or four times a day, with squill, ipecacuanha, or colchium, and some narcotic, according to the general state of the system and the prevalence of particular symptoms.

I need not detain you about some other bronchial discharges of rare occurrence. Bronchial hemorrhage may occur to a considerable amount, independent of any permanent disease ; and the fluid blood in the tubes will occasion a mucous rhonchus. In character it resembles epistaxis, hemorrhoidal flux, and other bloody discharges from mucous membranes ; but in this case, besides the weakness resulting from loss of blood, when this is considerable, there may be some danger from the interruption which it might cause to the function of respiration. The treatment, as in other hemorrhages, will depend on whether the hemorrhage is sthenic or asthenic—active or passive. These points are fully treated of by authors.

LECTURE XIV.

Diseases of the Air-tubes (continued)—Altered Sensibility or Mobility—Spasmodic Asthma ; Pathology and Course ; Physical Signs ; Extent and Effects of the Bronchial Spasm—Paralytic or Atonic Asthma ; Mode of Production and Signs—Treatment of Spasmodic Asthma ; Treatment of the Paroxysm ; General Treatment ; Signs of Improvement ; Treatment of Paralytic or Atonic Asthma—Treatment of Increased Sensibility of the Bronchial Membrane.

WE have surveyed in outline the features of those affections of the air-tubes which are either inflammatory or characterised by a change of their secretion. But besides such affections, there may be diseases of the *sensitive* and *moving* functions of these tubes. There may be an excess or a diminution of their sensibility, and of the contractility of their muscular fibres, which may vary with the sensibility or independently of it. We have already noticed these modifications of properties as producing certain varieties of cough ; excessive contraction of the bronchi giving the wheezing and whooping characters to cough, and their defective contraction occasioning it to be hollow, sonorous, and without expectoration. We have now to advert to the same modifications extending to the ordinary acts of breathing, and constituting what is commonly called *nervous* or *spasmodic asthma*. But from what I have just said, you may perceive that such disorders may be of very opposite kinds, and that the term *spasmodic* asthma will apply only to those cases in which the sensibility or contractility of the air-tubes, or both, are exalted, and that there may be another class of cases depending on a diminution of these properties, and to which may be applied the name *relaxed* or *paralytic* asthma. We have both spasmodic and paralytic affections of other muscular organs and canals—of the bladder and urethra, the intestines, &c. ; and analogy favours the supposition that we may have similar conditions of the muscular fibres of the air-tubes. But this subject has not yet been duly investigated ; and although I have seen many facts which support the view thus

presented by analogy, further research both at the bed-side and in the dead-house will be required to establish it in a practicable manner. That very able physician Dr. W. Stokes, has drawn the attention of the profession to this subject, in connexion with dilatation of the air-cells ; and if I mistake not, we shall do well to think of it in connexion with the functions of the tubes also. Let us first, however, attend to the better known complaint, spasmodic asthma.

The term asthma is generally given to dyspnœa occurring in paroxysms ; but we have seen that attacks of bronchial congestion, and bronchial flux, or dry and pituitous catarrhs, may come on suddenly, last a longer or shorter period, and cease in such a manner as to merit the name asthma ; by which term, in fact, they are generally known in this country. In the greater number of cases of asthma there is good reason to suppose that one or other of these affections, or some degree of inflammation, is present, and by increasing the irritability or the irritation of the bronchi, causes an undue contraction of their circular fibres. An increased vascularity of the bronchial membrane may heighten its sensibility, and augment the contraction of those fibres that are in relation to it, and the same result may ensue from the irritation of an unusual quantity or quality of secretion within these tubes. So, on the other hand, the continuance of inflammation, the thickened and altered condition of the membranes which it induces, may tend to impair their sensibility, and to injure in proportion the contractility of the tubes. In all these cases the modification of the sensibility and contractility of the air-tubes is secondary to other lesions which are more essentially vascular. But there are also cases of a purely nervous character, in which the disease is truly a neurosis ; and the temperament of the patient, the nature of the exciting causes, the very sudden attack and removal, and the irregular duration of the affection, sufficiently point out this character. Thus they commonly occur in nervous or hysterical persons. The attacks are excited by strong or peculiar odours, the smell of a stable, close rooms, particular conditions of the atmosphere, irritations of the stomach, or mental emotions ; and these causes often quite suddenly bring on the attack, which if severe, obliges the patient to assume a remarkable and very characteristic attitude, with the body bowed forwards, the arms resting on the knees, the chest contracted, with the feeling of a tight cord or heavy weight on it, the face suffused with an expression of great distress, the veins turgid, and perspiration soon beginning to flow freely ; whilst all the muscles of respiration, ordinary and supplementary, are trying their utmost to introduce air into the chest. With what success these efforts are made, we may learn by applying our ear to the chest, where in spite of the force of the motion, scarcely any sound of passing air is heard. The contractions of the muscles often give an external muscular sound ; but within the chest there is only a very weak respiratory murmur, with occasional wheezings or whistlings. The violent action of the muscles of inspiration seems to diminish rather than to increase the

entry of air ; when the efforts are less violent, especially towards the end of the paroxysms, now and then the air will be heard to enter quite well, as if the obstacle were suddenly removed, but at the next breath all is as obscure as before. At these instants we must suppose that the spasm of the bronchial muscles is momentarily relaxed, and Laennec has pointed out a method of causing at will this relaxation, which may be useful in enabling us to determine the nature of the disease. If we desire a patient, who labours under asthmatic spasm, to restrain his efforts of breathing, and to hold his breath altogether for a few seconds, or what amounts to the same thing, to count with his voice as many numbers as he can without taking breath, and then as quietly as possible to breathe again, the air will then be heard to enter freely into every part of the lungs, but in a breath or two after the spasm regains its hold, and the respiration becomes as obscure as ever.

Now how does this happen ? How does a moderate inspiration, *after holding the breath*, introduce air freely, when the strongest efforts are otherwise unavailing to produce the same effect ? Laennec used to say that the spasm was thus overcome *by surprise* ! This explanation is too poetical to be received in physiology. A surprise of the bronchial muscles conveys no definite idea to us, unless we suppose the tissues to be endowed with the mental faculty of *marvellousness*. I do not see how we can explain the phenomenon in question under any other supposition than that there is, as Laennec supposed, a temporary relaxation of a tonic spasm of muscular fibres ; and this relaxation I should ascribe to an increased degree of the same cause, which usually and naturally effects the relaxation of these fibres. We have before dwelt on this point in speaking of the physiology of respiration. We were then led to consider the contraction of the circular fibres, excited by a certain degree of foulness of the air within them, as an essential part of normal expiration. Now the foulness of the air being increased by holding the breath long, would stimulate these fibres to their utmost contraction—a contraction even beyond the state of asthmatic spasm : their irritability is thereby for the moment exhausted and the spasm becomes consequently relaxed, and the air is heard to enter freely ; but after a few moments' relaxation the irritability is again restored, and the exciting cause of the spasm remaining the next breath may find the contraction as strong as ever.

Such I believe to be the true explanation of the phenomenon in question ; and, did time admit, I should like to illustrate the principle on which it is founded by several examples of other spasmodic diseases, for it is one of much practical interest, and has not been sufficiently recognised. It is in this way that electricity will sometimes relax a spasm ; it stimulates a muscle to a contraction still more forcible than that of the spasm ; the irritability of the muscle is thus, as far as relates to the exciting cause of the spasm, exhausted ; and a continued application of the same stimulus may remove the excessive irritability, or subvert the morbid relations that habit has established between the contraction of the affected muscle and some

irritating cause in the system. Some of you have heard Sir Benjamin Brodie, on the same principle, explain how a spasmodic constriction in the urethra may be relaxed by the application of a bougie: the stricture refuses it passage at first; but by keeping the end of the bougie in contact with the stricture for a few minutes, the irritability of the contractile fibres is exhausted, the spasm is relaxed, and the bougie passes.

The distinctive physical sign, then, of spasmodic asthma is this imperfect sound of the respiratory murmur, even with forcible breathing, *except after holding the breath*, when it becomes as loud as, or louder than, usual. When the bronchial spasm is considerable, especially during the paroxysms, the chest sounds ill on percussion, not with the absolutely dull *mat* sound produced when solid or liquid is in the chest, but a short, tight, unresonant sound, like that which the chest yields on a forced expiration. This is caused by the contracted state of the lungs when under the influence of the bronchial spasm; the walls of the chest, therefore, being pressed inwards by atmospheric pressure, are not so free to vibrate as usual, when there is more of a balance of pressure on either side. You may generally obtain a better sound by striking on a finger or pleximeter pressed on the chest strongly enough to exceed the contraction of the lungs; this restores to them their springing resistant quality, by which they give a regularity to the vibrations of the thoracic walls. The same contraction of the lungs, when excessive, sometimes causes the diaphragm to rise higher than usual in the chest, and to produce a remarkable hollow at the epigastrium; and the whole chest presents a tight and contracted appearance.

Now you must perceive from all these signs that the contraction or spasm of the air-tubes must be very general, and extend to their very terminations; for spasmodic constriction of the large and middle-sized tubes cannot be complete on account of their cartilages; and with partial obstruction of these, the smaller ones and their terminations being free to expand, you would have the respiratory murmur more noisy than usual, and accompanied with all sorts of dry rhonchi, produced by the resisted passage of the air to these expansible parts, as we find in the first stage of bronchitis. But in spasmodic asthma there is very little sound, and so little expansion of the chest, that we are constrained to suppose that the smallest tubes are constricted likewise, and that either from their almost total occlusion, or from the presence of spasm in their very terminations, the vesicular structure itself is but imperfectly expanded. It may occur to you that I am assuming largely, in attributing these great effects to the operation of the tiny circular fibres of the air-tubes, whose muscularity has been questioned by many, and whose very existence in the smallest tubes is a matter rather of analogy than of demonstration. Well, I admit that we want further information respecting the physiology of the lungs, before we can put forth these views with confidence; but assuming that the whole system of the air-tubes does possess a power of muscular contrac-

tility (which I do on the authority of Vernier and Wedemeyer, who saw the smaller tubes contract almost to obliteration on the application of a mechanical or chemical stimulus), it is plain that a very minute force, contracting the caliber of each tube, will be enough to countervail a great force exerted by the muscles of inspiration. Recollect that the muscles of inspiration expand the lungs only indirectly, by atmospheric pressure; that this atmospheric pressure amounts to a weight of fifteen pounds to the square inch; that this amount of pressure is never realized by the power of the muscles; and that which is exerted, when distributed over the vast bronchial surface, becomes very small in each tube,—and you will be able to understand that the bronchial muscles, delicate as they are, may be sufficient to command the passage of the individual tubes, and to resist collectively the full introduction of air into the lungs.

Those who suffer much from spasmodic asthma are seldom free from a shortness of breathing in the intervals; and the frequent recurrence of the paroxysms generally brings with it more of this habitual dyspnoea. If we examine their chests we find the same diminution of respiratory sound as during the paroxysm, but in a less marked degree; and the test of holding the breath proves that spasm exists here also, having become in a measure habitual. No doubt the frequent recurrence, or long continuance of these spasmodic contractions of the tubes, must lead to a permanent diminution of their caliber, and the other tissues change and fix them in this constricted size. We see the parallel of this in the irritable bladder, which, after long continued attacks of spasm, eventually becomes permanently contracted. Where the disease is purely spasmodic, this more lasting change might not ensue for a very long period: but I have before mentioned to you, that with spasm of the circular fibres there is so commonly associated congestion, irritation, or inflammation, or these are so frequently induced by the spasm, that the phenomena of these pathological conditions are very commonly combined with those of spasmodic asthma. Hence, in asthmatic subjects, you may have an attack of bronchitis, of dry catarrh, or of humid catarrh, assume in its course a spasmodic character; and you may have a paroxysm of asthma, which came on suddenly as a spasm, terminate by a copious catarrhal secretion. The latter seems to be a common course of the asthmatic paroxysms, which are sometimes associated with organic diseases of the heart. The congestion which these determine in the membranes and structures of the lungs exalts their sensibility and irritability; and where the circular fibres are naturally disposed to spasm, this most readily excites it; and this spasm may not be entirely relaxed until the congestion is relieved by a free secretion from the bronchial membrane.

Hitherto we have considered only the spasmodic form of nervous asthma, or that dependent on an *excessive* sensibility and contractility of the bronchial tubes; but as, in examining the elements of dyspnoea, we found that a *defect* of these properties would disorder

the process of breathing, we are led to inquire whether there may not be a nervous asthma or dyspnœa of this kind, from weakness, or a *paralysis* of the circular fibres, or of the nerves, whose sensibility guides their contractions? We have parallel affections of the alimentary and urinary passages, when, from local or general causes, their moving fibres become torpid or paralyzed: and if I am right in supposing that the action of the circular fibres, and the elasticity of the longitudinal fibres of the bronchi, are essential to the effectual performance of the act of expiration, defects of the properties of these tissues must cause a proportionate imperfection in this act. There are some facts which seem to me to bear directly on this point. I mentioned to you before, that Mr. Swan found, that in animals in which the par vagum had been divided in the neck, the act of inspiration became imperfect, and the lungs permanently distended. Laennec remarked, that the lungs of persons who died from suffocation in sewers, appeared, on opening the bodies, uncommonly distended, and did not collapse as usual; and he proposes it as a query, whether this may not be from a sudden dilatation of the air-cells. Now, sulphuretted hydrogen gas, which is a chief component of the exhalations of sewers, is remarkable for its power to destroy muscular irritability; and it seems very probable that its direct paralyzing effect on the bronchial fibres may be the cause of the distension remarked by Laennec. We shall have occasion to return to this subject, in connexion with the lesion *dilatation of the air-cells*, termed by Laennec, *emphysema of the lungs*; and we shall then advert to the view of Dr. W. Stokes, which embraces a similar pathological notion. The phenomena of impaired contractility of the bronchial fibres would be the converse of those of spasmodic asthma; the chief difficulty being in the act of expiration, which would be performed imperfectly and with much wheezing and effort, whilst inspiration might be short and comparatively unembarrassed. Now we do meet with many instances of dyspnœa, generally with old bronchitic affections, but occasionally in cases more of a generally nervous character, and in hysterical females, in which this difficulty of expiration is the prominent feature. So, also, we see the defective action of the contractile fibres of the intestinal tube arise sometimes from previous over-irritation, and sometimes from more directly weakening or paralyzing causes, and sometimes from that irregular distribution of the nervous influence that produces the phenomena commonly called hysterical. Nay, if we consider that irritations and inflammations first exalt, and afterwards injure the contractile properties of hollow organs or tubes, and that these irritations or inflammations affect successively different parts of the same tubes, we can see that spasmodic and relaxed asthma may co-exist in the same person, one part of the bronchial tubes being unduly contracted, and another unduly relaxed, from an irregular distribution of the property of irritability. We have not time to pursue this subject further at present, but we may revert to it by and by in connexion with dilated bronchi and pul-

monary emphysema; and, in the meantime, I recommend you to bear it in mind when cases of asthma or dyspnœa come under your notice.

The treatment of spasmodic asthma may be considered in relation to the paroxysm, and to the general state of the body in the intervals. One point is to counteract the exciting cause of the spasm; the other, to remove or destroy this cause altogether. To relax the spasm of the bronchial tubes, various measures may be suited, according to the immediate cause of the spasm. When this is purely nervous, with little or no bronchitic or catarrhal complication, such antispasmodics as æther, valerian, asafoetida, opium, belladonna, and the fumes of stramonium or tobacco, will sometimes succeed, and each one of these has proved more successful than the others in particular cases, but seldom retains its efficacy long. A more generally and permanently successful remedy is strong coffee, made by infusion, long ago recommended in this country by Dr. Bree, and much extolled by Laennec. I have known some asthmatic persons who relied so much on its efficacy, that the very idea of being out of the reach of it would be enough to bring on a fit; and they most scrupulously avoided using coffee as an ordinary beverage, lest this should impair its efficacy as a remedial agent. This was a good rule, for even this is not free from the tendency of antispasmodic and narcotic remedies in general, to lose their power by frequent repetition. In some cases, sudden strong impressions on the system, such as by dashing pails of cold water on the body, or passing moderate electric shocks through it, have been known to stop a paroxysm of asthma; and their mode of action must be referred to the principle of which I have spoken before. I know of one remarkable case, on which there is apt to come on quite suddenly a loss of voice, and even of the power of articulation, from a spasm of certain laryngeal muscles, and to some degree of the bronchi also. The subject is a lady of strong mind, and by no means fanciful or (according to the general application of the term) hysterical. For a considerable time relief was instantaneously given merely by her taking a few electric sparks with her fingers. This remedy afterwards lost its efficacy; and even shocks failed to relax the spasm. Subsequently it was found that holding a lump of ice in the back part of the mouth was as effectual in loosening the tongue and the breath as the electric spark had been. This affection was purely nervous; it could be excited at any time by a strong mental emotion, or a slap on the back, and always ceased as suddenly as it came on.

If the asthmatic spasm be complicated with bronchitic or catarrhal affections, which is very frequently the case, the remedies recommended for these may often be advantageously combined with some of those just named; and when the nervous affection does not form the chief part of the complaint, it is probably dependent only on the altered condition of the membrane, which is either inflamed or congested, and to which, therefore, the remedial agents must be chiefly addressed.

The fulfilling of the second indication, to diminish excessive irritability of the bronchial muscles, or to remove the causes of irritation by which they are excited, will be best aimed at by various means which tend to restore a proper balance of the functions of the whole system, and to improve its general health. Of these the most effectual are those of diet and regimen. Particular rules can scarcely be laid down, as the proper measures will vary greatly according to the circumstances ; but the experience of the patient will generally give a clue to the most eligible plan. The shower-bath, and moderate exercise in the open air, avoiding walking against a strong wind, are very beneficial in most cases. Of medicinal agents, besides those necessary to regulate the secretions, the metallic tonics sometimes do good, by diminishing the morbid mobility of the bronchial muscles, or the sensibility of the nerves that influence them ; and I have known, in various instances, the sulphate and oxyde of zinc, the subnitrate of bismuth, the nitrate of silver, and the sulphate of copper, severally beneficial in diminishing the tendency to the recurrence of the paroxysms. Probably these remedies act through the nerves of the stomach (the par vagum), which are so closely associated with those which influence the bronchial fibres ; and they may do this directly, or indirectly, by improving the condition and function of the stomach, disorders of which, in some form or other, are so commonly associated with spasmodic asthma. It has often occurred to me that *lead*, which is known to exert such a paralyzing influence on the muscles of both animal and organic life, might be capable of lowering their irritability when excessive and excited to spasm ; but its administration would require much judgment, and I have not had an opportunity of trying it.

The signs of improvement are (besides the less frequent occurrence, diminished severity, and shorter duration of the paroxysms), a freer state of the respiration in the intervals, so that the vesicular murmur is pretty audible, without much admixture, throughout the chest, and is increased in loudness by quicker and deeper inspirations, not stopped or impaired as during the continuance of the asthmatic tendency, when additional effort will often at any time excite the spasm. In the cure of this, as of other spasmodic disorders, it is very necessary to watch the circumstances that excite the paroxysms, in order to be able to avoid them ; for the frequent occurrence of spasm increases the chance of its recurrence, until it becomes habitual, and may be excited under almost any circumstances. The evil of an habitual asthma is not only the inconvenience and distress occasioned by the paroxysm itself, but also the permanent changes which it may induce in the structures of the lung—such as contraction and rigidity of the air-tubes, congestions and other lesions of the parenchyma, diseases of the heart, &c.

I have little to say on the treatment of the dyspnœa arising from paralysis, atony, or weakness of the contractile and elastic fibres of the air-tubes. Depending, as this affection usually does, on previous inflammatory lesions, the remedies generally useful at the decline

of those lesions, are such as may be supposed to act, in some measure, by stimulating or giving tone to the bronchial fibres. Thus ammoniacum, myrrh, benzoin, the balsams of copaiba and Peru, and the inhalation of tar and other stimulating vapours, besides their operation on the discerning function of the air-tubes, may probably have an influence of this kind on their moving fibres : and they may thus improve their condition, in relation to the act of both expiration and expectoration ; the difficulty of which often forms the most prominent feature in many protracted cases of inflammatory and congestive disorders of the air-tubes.

Most commonly, when the sensibility of the bronchial tubes is increased, their contractility is so likewise ; when the inhalation of cold air is painful, it also seems to take away the breath : but I have before hinted, that the animal sensibility and organic contractility may not always be in proportion to each other. Thus spasmodic asthma may be unattended with any other pain than that common to dyspnœa ; and the relaxed state of the bronchial tubes, marked by difficult or imperfect expectoration, is sometimes accompanied by an increased sensibility of the bronchial membrane, so that the breathing of cold or irritating air becomes unusually painful. In a few such cases, I have known relief to be afforded by the inhalation, twice or three times a day, of the vapour of hot water, to which a few grains of camphor have been added. When this fails, I should be disposed to try other narcotics, such as hyoscyamus, or conium ; which may be diffused through the water in the form of recent extract, or saturated tincture, rendered more volatile by a small addition of liquor potassæ. The proportions must be determined by experience, beginning with small quantities. In these cases, and others in which soreness, or rawness of the stomach is complained of, much comfort may be derived from the use of Jeffreys' *Respirator* ; or if the patient do not choose to be so muzzled, some of the same benefit may be obtained by tying a porous silk handkerchief, or a piece of fur, over the mouth ; or, in the case of ladies, even by wearing a thick veil.

LECTURE XV.

Diseases of the Air-tubes (concluded).—Changes of structure : Thickening of the Membranes ; Hypertrophy of the Longitudinal Fibres ; Rigidity ; Pathology and Signs ; Treatment.—Dilatations of the Bronchi ; Varieties ; Pathology and Mode of Production ; Symptoms and Effects ; Physical Signs and their Distinction from those of Phthisis ; Amphoric Sound on Percussion ; Treatment and Prevention.—Ulcers of the Bronchi.

WE have now to notice certain changes of structure which the air-tubes occasionally present. These are, I believe, generally the con-

sequences either of inflammation, or of some kindred modification of the vascular function affecting the nutritive process. Inflammation, when often recurring, or long continued in the bronchial membranes, as elsewhere, induces a change of structure ; and the mechanical forces to which they are subjected in the function of respiration may modify this change in various manners.

The most simple change of structure is a mere thickening of the mucous and the submucous membranes, which you see exhibited in these drawings. This generally in some degree, accompanies acute inflammation ; but it is then only temporary, and subsides as the secretion becomes free and albuminous, being caused, probably, by only an infiltration of the pores of the tissues with soft lymph, which as the inflammation subsides, is eliminated and expectorated with the mucus of the membrane. The deposits that are the most readily produced by inflammation in highly vital tissues, are also the most readily removed ; and thus it is, that the soft albuminous matter that is effused by acute inflammation in cellular textures, and in parenchymata in general, if it be not so abundant as to interfere with the absorbent functions of the vessels, becomes absorbed as the inflammation subsides.

But it is otherwise when the inflammation recurs frequently, or is of long duration ; for it then causes an effusion of a less absorbable nature, involves the less vital structures, and as the changes induced are slow, so they are more permanent, because they become identified with the nutritive or reparative functions of these tissues. There will then be produced a degree of hypertrophy of some or all of the various tissues composing the tubes. Sometimes there will be an extraordinary growth of the mucous membrane, such as appears to be represented in this plate of Dr. Carswell's, where there is also a great dilatation of the tubes. More commonly, however, it is the harder and less vital textures, that undergo the change, and its effect is to increase the rigidity of the tubes, so that there is a diminution of their expansibility and contractility. Nothing is more common than to see the air-tubes of persons who have long suffered from bronchitis, presenting an undue development of the longitudinal elastic fibres ; whilst in other cases the outer cellular coat of the larger bronchi is thick and indurated, and their cartilages are sometimes partially ossified. Any of these changes has the effect of rendering the lungs less easily expandible in respiration ; the first in particular is a common cause of the short breath, which persons frequently effected with bronchitis generally manifest ; and although not often serious in itself, yet it may so abridge the sphere of the function of respiration as to make its increased exertion, on bodily exercise, a matter of difficulty and disorder, and to render it ill able to bear any other attacks of disease, to which the lungs can in general adapt themselves by supplementary efforts. Thus when one portion of a healthy lung is attacked with pneumonia, or compressed by a pleuritic effusion, its function

is supplied by the increased and quickened movements of the other portions, which, in their natural state, are equal to this augmented task ; but if their pliant elasticity be impaired, and their size more fixed by an increased stiffness, they will be, in proportion, less available for additional exertion, and the body will suffer the more from the crippled state of the function.

The chief sign of hypertrophy of the longitudinal fibres, and of increased rigidity of the tubes generally, is, *difficulty of inspiration*, which is short, quick, and performed with an effort, especially on making any exertion ; whilst the expiration is comparatively easy ; but both acts are often accompanied by wheezing sounds, from irregularities in the caliber of some of the tubes, and frequently from partial congestions or inflammation, from which tubes thus diseased are rarely free. The vesicular murmur is impaired, and the expansion of the whole chest is perceptibly limited. These signs resemble those of spasmodic asthma, except that they are permanent and are not removed as the latter may be for an instant, on breathing after holding the breath, as I described to you in the last lecture.

Inasmuch as these lesions seem to arise from continued inflammation, it becomes of the more importance to direct remedies against those forms of bronchitis that are habitual, or frequently recurring. An imperfectly cured cough will often hang on a patient for months and even for years. Not being much incommoded by it, and the general health not suffering materially, he will not think it worth while to persevere in the use of remedies, or of a proper regimen. In the process of time, however, especially under the influence of fresh colds, to which he is always more liable, the breathing becomes permanently shortened, and an irritation is often fixed in some of the affected tubes, and manifests its effect on their secreting function by an habitual expectoration, generally of a thin mucous, or pituitous character. This affection varies greatly in degree. I have seen several cases of severe habitual dyspnoea, which ultimately proved fatal, present all the characters which I have described ; and there was found, after death, no other lesion than a general redness of the membrane lining the larger tubes, and an extraordinary development of the longitudinal fibres. This appearance is very common in the bodies of old people who have long been subject to cough and shortness of breath ; but I have seen it also in the middle aged, and in a few instances in younger subjects. It is not, however, met with in all cases of protracted bronchitis ; these sometimes lead to other and opposite results ; and it is probably connected especially with some kinds of inflammation, which as in other situations, show a particular disposition to affect the fibrous tissues ; but further observations are wanted on the subject. There is one point with regard to treatment, which is suggested by a knowledge of this change of structure—that not only should we persevere in the use of the means which tend to eradicate the low degrees of inflamma-

tion that produce it, especially alkaline expectorants and counter-irritants, but we should also endeavour to countervail, by mechanical means, that mechanical limitation which this change induces in the size of the tubes. If the patient use no exertion, and give his lungs little play, any increase in the rigidity of the tubes will more readily fix them in their present contracted state : but if he take moderate exercise, increased as habit improves his power, the lungs will be kept in that free mobile condition that is least favourable to rigidity or deposition of any kind. Probably even special efforts of inhalation would be useful with the same view ; and as this might be combined with some mildly stimulating vapour, such as that of water impregnated with tar, camphor, or the like, it might be made also serviceable in improving the condition of the secreting membrane. You can readily perceive, however, that great discretion is necessary in the employment of these mechanical means ; for if they strain the tubes beyond the due limits, they may cause a morbid yielding of their walls, and increased inflammation ; and if exertion be used beyond what the function of respiration can support, it will occasion congestions in the lungs, which may aggravate the original disease, and may induce lesions of other kinds. They are more eligible in young than in old subjects, for in the latter the change is more likely to be permanent, under the influence of that general law by which, as age advances, fibrous tissues tend to assume a cartilaginous hardness, and cartilage becomes rigid with osseous matter.

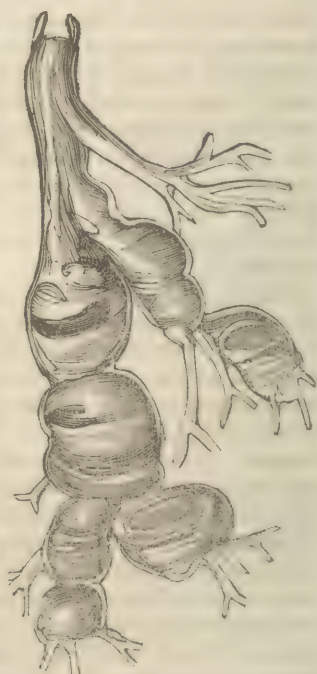
There is another kind of alteration of the air-tubes that has attracted more attention than those which I have hitherto described. I mean *dilatation of the bronchi*. This affection deserves attention, not only on its own account, but also because it sometimes produces physical signs which closely resemble those of phthisis. The drawings before you exhibit different forms which the dilatations assume. To show them well, the bronchi should be laid open, from their large to their small branches, with a pair of scissors ; and in examining lungs in this way, it is not at all uncommon to find the calibers of the branches larger than those of the trunks from which they proceed, the enlargement being generally most manifest in those parts of the tubes where the cartilaginous plates are small and few ; but occasionally the larger tubes are dilated also, their rings only here and there limiting the dilatation. Sometime the dilatations are pretty uniform through some length of a tube (as in fig. 1). In other cases they form irregular roundish cells or pouches freely communicating with each other, and from which tubes of unchanged size here and there arise (fig. 2). The tissues composing the tubes are generally, at the same time, more or less altered. They are least so in the tubular form of dilatation (fig. 1), in which the coats are often quite thin, and the longitudinal fibres are distinct, although occasionally enlarged. But in the more globular dilatations, the walls of the tubes are generally much altered. They are irregularly

thickened ; the thickening being formed in part by hypertrophy of the mucous or submucous tissues lining the cells, which sometimes form folds of wrinkles around the tubes, and partly by a dense tissue on their outsides, probably consisting of the parenchyma of the lung compressed by the encroaching tube. There is little or no trace of the longitudinal or circular fibres in this form of dilatation, and the lining membrane is generally in a softened state, and of a red colour, whilst there may be considerable rigidity in some parts of the tubes. This is the worst kind of dilatation, as you may suppose, from its more complicated character : but you will be better able to understand this if we examine a little further into the causes and pathology of dilatation of the air-tubes.

Fig. 1.



Fig. 2.



Laennec, who first described these lesions, attributed them to the frequent accumulation of mucus in the tubes, causing their mechanical distension. He considered that they were formed especially by long-continued chronic bronchitis, and that the continual recurrence of the same distension of the tubes led to their permanent dilatation. But this view has always appeared to me to be quite insufficient to account for the remarkable changes which we frequently see in the

structures of the dilated tubes ; and, according to my observation, these lesions do not by any means constantly occur where the bronchial secretion is copious, and most calculated to cause distension. Neither do we in chronic bronchitis often meet with such a complete suspension of the respiratory murmur as these supposed distensions with mucus ought to produce. The expectoration is generally more diffuent, and less likely to accumulate in the tubes, than that of dry catarrh, and the latter stages of acute bronchitis. M. Andral takes a more rational view of these lesions, in ascribing them to a modification of the nutrition of the textures composing the tubes ; but he does not attempt to give any specific explanation of the mode in which their form becomes so remarkably altered.

If you bear in mind all the circumstances of the mechanism of respiration, as we have been considering them in this course, and the various modes in which they may be deranged by the effect of disease on the textures which form parts of it, you will soon find no difficulty in accounting, in several ways, for the dilatations of the air-tubes, as well as for the modifications of nutrition which accompany them, and their effects on the adjoining tissues. I have had occasion to point out to you more than once that a mutual pressure is continually exerted between the interior of the bronchial tree and the air admitted into it by respiration ; in inspiration, by the air which enters to distend the tubes ; in expiration, by the tubes contracting to expel the air. In forcible acts of respiration, such as coughing, or energetic breathing, this pressure is increased ; but in the normal condition of the tubes, when they all convey the air equally and freely to and fro, and meet the pressure with a well-proportioned degree of elasticity and contractility, this pressure is balanced and borne well ; and instead of causing disturbance, it serves to keep the air in a constant relation to the blood, and to regulate the circulation through the lungs. But disturb in any way the equality of this pressure, or derange those elastic and contractile properties which are opposed to it, and you may then convert it into a cause of unnatural distension in some parts, whilst it does not reach others with sufficient force. Now there are several circumstances that may cause these disturbances, and they are especially to be met with in those diseases which are known to lead to dilatation of the air-tubes. Bronchitis may act in both of these ways. By thickening of the membranes or viscid secretions it may cause partial or complete obstructions, which by preventing the free entry of air into some tubes, give increased force to its pressure in others, which become distended in consequence ; and it may so alter the condition of the tissues composing the tubes, that, losing their elastic and contractile properties, they yield to the pressure and become fixed in this dilated condition. Perhaps, as Dr. W. Stokes has suggested, the mere loss of contractility may be sufficient in itself to cause dilatation of the bronchi ; but I think that our view will be more complete if we take into account other circumstances which we know to be often present, and the operation of which is perfectly intelligible. Just

Fig. 3.



glance at this sketch (fig. 3), and you will see at once how an obstruction (*a*) preventing the air in inspiration from entering one set of tubes (*b*), will cause the excessive distension of the adjoining tubes (*c c*); and recollect that this is taking place in tubes softened or otherwise modified by inflammation, and you will perceive how the dilatations may become perpetuated, and liable to increase through the altered condition of the constituent textures. Hence the lesions are often not simply dilatations of the tubes, but comprehend also irregular softening and indurations, absence and thickenings of their several textures; so that when the lung is cut open after death, it may be at first difficult to distinguish that the irregular cavities which it presents are formed by dilated tubes. Then in the production of these dilatations we are not to forget the influence of violence in the acts of respiration. They have been observed especially to succeed to whooping-cough and other bronchial affections in which the cough is particularly violent and long-continued.

The effects of these violent acts of breathing may be twofold; they may cause the dilatations by the prolonged and forcible inspiration in the manner already described; and they may increase them irregularly when so distended, by the sudden pressure of the expiratory forces upon them. But I have met with cases of dilated bronchi in which there had been very little cough, and none of any violence; and here we must suppose that the other conditions, the irregular introduction of air and the yielding of the membranes, were more exclusively concerned in the production of the lesion. I will tell you of another manner in which the bronchi become dilated. In the disease called pleuro-pneumonia the lung is inflamed, and at the same time compressed by an effusion in the sac of the pleura. Now if it remain long in this state, the smaller air-tubes and cells become obliterated by the adhesion of their sides, so that when the liquid is removed from the pleura they will not expand again with the enlargement of the chest; but the large and middle-sized bronchi are not obliterated; they bear the whole force of the inspired air, and become consequently dilated by it. This kind of dilatation is usually conjoined with contraction of the affected side: we shall notice these cases hereafter; they are not very uncommon, although they are not to my knowledge noticed by any writer. Any other circumstance which causes the obliteration

tion or obstruction of a considerable number of the bronchial tubes and cells, must tend to produce a dilatation of the adjoining tubes on which the motions of the chest would act with augmented force.

The symptoms produced by dilatations of the bronchi will be according to the extent of the lesion. Slight degrees of it are met with in the bodies of persons who had not during life manifested any prominent disorder of the respiration; and its simpler forms may exist to a greater extent without producing other effect than a liability to attacks of bronchitis. But where it affects many tubes, has modified their structure, and has enlarged them to such an amount that they press on and obliterate a considerable extent of the pulmonary parenchyma, it then causes habitual dyspnœa, with more or less cough and muco-purulent expectoration, which is often remarkable for its fœtor. There are, generally, present also the ordinary symptoms of severe chronic bronchitis, from which some parts of the affected tubes are scarcely ever free; and the permanency of these symptoms, together with a degree of lividity, dropsical effusion, and cachectic condition, often induced by the crippled condition of the lungs, forms the usual general character of the aggravated forms of dilated bronchi.

Now you may say that these symptoms look very like those of consumptive disease; and so they well may, for there is injury to the function of respiration, profuse expectoration, hectic fever, and the patient is often slowly wasted away. In their aggravated forms, dilated bronchi are not more tractable than tubercular consumption itself; but their tendencies and constitutional effects are different, and merit as much of a distinction as our means of diagnosis can find for them. Unfortunately, this is not one of a very marked kind; in fact, in many instances it is to be made only by those much experienced in diagnosis; and even they will give it in terms rather of probability than of certainty.

You can readily understand that the air passing in bronchi dilated to a large size, or into cavities, will give a hollower, a more blowing sound, than in those of the natural dimensions; hence, over them the sound of respiration may be bronchial, tracheal, or cavernous, in regions where it is naturally purely vesicular; and if, as it frequently happens, there be liquid in the tubes, the bubbling into which it is thrown will be heard to be coarse and gurgling, instead of the finer mucous rhonchus of common bronchitis. So, also, the voice may be powerfully transmitted through these enlarged tubes; not in a diffused fremitus, as usual, but loud, and startling, as if issuing from the spot; in some cases cracked and jarring, as in bronchophony; in others more articulate, and with a snuffling and hollow sound, as in pectoriloquy. But these are also the signs of tubercles and excavations in consumption; and we must seek for further distinctions. Besides, in the history of the case, and the character of the constitution, these distinctions are sometimes to be found;—in the situation of these sounds, which, in phthisis, is usually in the superior parts, but in dilated bronchi in the middle

regions of the chest; in their character in relation to time, those in phthisis tending to increase and spread as the excavations proceed, whilst those of dilated bronchi remain nearly stationary for weeks and months; in their being less change in the shape of the chest with dilated bronchi than with phthisis, unless they have arisen from pleuro-pneumonia, in which case the change is different; and, finally, in the nature of the sound on percussion, which, in phthisis, is more extensively dull, especially under the clavicles, whereas, in dilated bronchi, if any dulness exist, it is generally in the mammary, lateral, or scapular regions of the chest, and is often accompanied by a sound of a peculiar kind. This is a hollow tube-like sound, and from its resemblance to that produced by mediate percussion on the trachea, or by tapping with the finger on the mouth of a small phial, I have given it the name of *tracheal* or *amphoric*. I can give you a notion of the kind of sound, by filliping on a finger pressed on the larynx or trachea, or on the cheek when the mouth is opened in the manner of sounding the letter O, thus—. This sound depends, not essentially on the vibration of the walls, as in the case of the ordinary sounds of striking the chest, but on that of the air in the tubes or cavities, which give a note according to their length and size, precisely in the manner of a pan-pipe, or of this India-rubber bottle. In the natural condition of the chest you do not obtain this sound, because the stroke of percussion, and the resonance of the large tubes, are intercepted by the ill-conducting tissue of the lung: but when the bronchi are dilated so as to reach nearly to the surface, or, as we shall hereafter see, more perfectly when the large tubes are brought in contact with the walls of the chest by the pressure of a liquid effusion; or when, by perfect hepatization, the stroke and the resonance can be transmitted to and from these tubes near the root of the lung, you will then get various degrees of this amphoric sound, which it is not difficult to distinguish from the duller and deeper resonance of the healthy chest. The circumstances which favour the production of this sound are the same as those which cause morbid bronchophony and bronchial respiration, and pectoriloquy and cavernous respiration; but it requires a more perfect degree of them. Hence, although somewhat of the amphoric sound is occasionally yielded by empty cavities in phthisis, this does not often occur, it being damped by the irregularity of their form and materials, and the remains of spongy tissue on their surface. The cracked-jar sound (*bruit de pot fêlé* of Laennec) is of the same class, being a slight noise of a sudden motion of air and liquid within resonant tubes or cavities, produced by the impulse of external percussion. It is more common in large phthisical cavities, because their form and size more readily expose them to the influence of an impulse on the walls of the chest.

Finally, you will be better able to distinguish dilated bronchi from phthisical cavities, when you become fully acquainted with the signs and general symptoms of the latter; and I shall now only add, by way of recapitulation, when you meet with a case in which

long-continued cough, with purulent expectoration, dyspnoea, loss of flesh and strength, hectic fever, even with some of the physical signs of cavities in the lungs, beware of pronouncing it to be tubercular, if qualified by all or most of the following conditions:—If no proofs of a scrofulous habit can be traced; if the complaint have originated in a long-continued and violent cough, or in an attack of pleuro-pneumonia, and, considering its duration, emaciation have not proceeded very far; if the purulent expectoration have been foetid and sanious rather than flocculent or caseous; if the bronchial or cavernous respiration, voice, or gurgling, be heard rather in the middle than in the upper portions of the chest, and be there spread over a considerable extent of surface; if these middle portions chiefly sound differently on percussion, being dull when the rest of that side sounds pretty well, or amphoric when the side is generally dull and contracted; and if, although the cough and expectoration continue undiminished, these signs remain stationary for many weeks together.

I have little to say on the treatment of dilatations of the bronchi. You can perceive that when once formed, they can be little under the influence of medicine. The profuseness of the secretion may sometimes be restrained by acid mixtures; and I have known the nitro-muriatic acid in two or three instances succeed in removing its fœtor. Probably inhalations of chlorine would be useful in such cases. Where the cough is violent or troublesome, it should be allayed as much as possible by sedatives, such as hyoscyamus, belladonna, conium, and particularly opium, or some of the preparations of morphia, due attention being at the same time paid to the state of the excreting functions, and the general condition of the system, which may need various kinds of treatment in different cases. The co-existence of chronic bronchitis often renders external counter-irritation of some service in dilatations of the bronchi; and other antiphlogistic measures are occasionally required on the supervention of intercurrent acute inflammation, which sometimes takes place. It is, however, from preventive measures that we may expect more success; and our knowledge of the causes and tendencies of this lesion suggests the expediency of not abandoning the treatment of cases of bronchitis, pertussis, and pleuro-pneumonia, until all cough, and the physical signs, have been satisfactorily removed. Most of the severe cases of dilated bronchi that have fallen under my observation, I have traced to imperfect treatment in former inflammatory attacks; and I am fully convinced that many examples of the different structural changes of which I have been speaking in this lecture, may be prevented by an efficient plan of medication, when the complaint is considered merely as a severe cold, a cough, or “the influenza.”

I need not detain you on other structural lesions of the air-tubes, for they are of too rare occurrence to be of much practical importance. Ulcers of the bronchi seldom occur but in connexion with some cause which concentrates inflammation in the bronchial mem-

brane in a peculiar manner, such as the habitual inhalation of irritating particles of dust, in the occupations of needle-pointers, stone-masons, and leather-dressers ; the continued passage of tubercular matter in phthisis, and occasionally the specific influence of measles, small-pox, and syphilis. I do not know of any signs by which the presence of ulcers in the bronchi can be distinguished ; they rarely, if ever, exist without a similar affection of the larynx, in which case the voice is impaired or lost ; but this happens commonly when the bronchi are not ulcerated, or only so far as to give vent to the matter of vomicæ in phthisis.

LECTURE XVI.

Diseases of the Pleura ; Structure of the Pleura—Acute Pleurisy—Pathological History ; Symptoms : Physical Signs—Signs from Increased Sensibility ; from Friction ; from Effusion ; from Supplementary Action—Of the Sound Side—Modifications by Adhesions ; Columnar Adhesions ; Circumscribed Effusions ; Explanation of Anomalous Cases.

HAVING considered what may be called the ærial or mucous surface of the lungs, and the manner in which it is modified by disease, we now come to the other surface, and the pathological properties of the serous membrane which covers it, and lines the cavity in which the lungs move. This membrane in the healthy state is thin, almost transparent, and possessed of considerable elasticity, inasmuch as it remains smooth and unwrinkled, whether the lung be expanded or collapsed. It consists of two layers—one distinctly serous, which is always bedewed with a serous fluid, lines the cavity of the chest, and forms the outer covering of its organs. The other is clearly fibrous in the costal pleura, and, together with that of the pericardium, seems to be a continuation of the deep-seated cervical fascia. An under layer of the pulmonary pleura has also been described, I believe for the first time, by Dr. Stokes ; but whether this be, as he supposes, *fibrous*, or of the same structure as the outer serous layer, appears to me doubtful. It may be something intermediate between the two, for both serous and fibrous membranes consist of condensed cellular tissue, and vary only in condensation and softness, elasticity and rigidity, according to the office of the parts which they cover. The sensibility of the pleura is naturally very low ; and there is neither feeling nor irritation caused by this motion of the pulmonary on the costal pleura in respiration ; for a constant secretion of serum gives to both a smooth slippery surface, which reduces the friction to the least possible amount. Its blood-vessels are exceedingly fine ; indeed, it is denied by some anatomists that the serous membrane itself has any vessels, and they say that those which are visible are in the subserous tissue which unites it to the organ. I cannot

subscribe to this opinion, for I am sure that I have seen red vessels on the very surface of the pleura, when inflamed ; although these vessels probably conveyed only serum in the healthy state. No doubt the larger and more visible vessels do exclusively belong to the subserous cellular tissue, and we have before noticed that these are derived from both the pulmonary and the bronchial arteries.

It is chiefly these subserous vessels that form the striated patches or points of redness that are seen in the first stage of pleuritic inflammation, and their distension can be felt through the serous membrane, which feels slightly uneven on passing the finger over it. Probably at this period there is a diminution of the serous secretion at the inflamed spot, as we know such to be the first effect of inflammation in the mucous membranes : the effect of this would be to increase the friction between the surfaces. Very soon, however, the flow of serum is increased, and with it, if the inflammation continue, an albuminous matter, coagulable lymph, is also exuded by the serous membrane. This instance illustrates the simplest form of inflammation. We have the vessels with no compound structure to complicate or modify their action, and we find the increased development of these vessels by inflammation obviously attended by an exaggeration of those secretive functions which they fulfil in health. These functions are two-fold—that of liquid exhalation, and that of solid nutrition. The matter exhaled is serum ; the material of nutrition is the albuminous or fibrinous part of the blood. In their natural proportion, these functions preserve the membrane in a healthy state ; one merely lubricating its surface with a slightly albuminous fluid, the other nourishing and sustaining the solid matter of the membrane. When moderately increased by slightly augmented vascular action, these properties of the vessels cause hydrothorax and a simple hypertrophy or thickening of the subserous tissue, the most vascular part ; and this result we often meet with in an opacity, without adhesions, of the pulmonary pleura or other serous membranes. But when the exhalant and nutrient properties of the vessels are exalted by acute inflammation, there is as it were an overflow of their products ; the liquid effusion is rapid and copious ; and the excess of the nutritive secretion now appears on the exterior of the membrane in various forms, and either by itself, or mingled with the liquid effusion, constitutes all the different products which are recognized as the result of inflammatory action. In its smallest proportion it is held in solution by the effused liquid, which, on being drawn from the body, or after death, gelatinizes by this fibrinous matter which it contains. When very abundant, this forms films of lymph on the surface of the membrane ; and this lymph will generally be more abundant and disposed to speedy organization, when the inflammatory orgasm is strong, and the blood is rich in nutrient matter.

Following still the pathological history of pleurisy, we find in this same lymph the matter of adhesions ; but whether or not these adhesions shall take place, will depend on the quantity of liquid

effusion between the pleuræ. This effusion will, of course, to a certain degree ponderate to the lowest parts of the chest, and in those parts will keep the pleuræ separate; and if it be the upper portions of the pleura that are inflamed, they will the more readily adhere, unless the liquid effusion be very abundant. But if the pleuræ be inflamed only in their lower portions, a moderate quantity of liquid will be enough to keep them separate; and if the lymph then become organized, it forms, not an adhesion, but a false membrane coating of the lung, which may have further effects in modifying the remains or the products of the previous inflammation. Before we consider these various results of the modifying influence of time, of the degree and kind of inflammation, and of previous disease, on the further pathological history of pleuritic cases, we had better take a view of the symptoms and signs of the more simple and recent forms of pleurisy.

You know well enough the general symptoms of pleurisy, and how characteristic they sometimes are; the sharp cutting pain in the side restraining every common inspiration, and often making the act of coughing or deep breathing almost intolerable; the short breath which consequently results, and the lying on or holding the affected side; the short dry cough; the general inflammatory fever, hard quick pulse, heat of skin, flushed cheek, &c.; but I have had occasion to tell you that extensive pleurisy and its consequences, may be present without any of this array of symptoms; that even in cases in which there had been scarcely a suspicion of the presence of disease in the chest, acute inflammation, and its concomitant, copious effusion, had been for many days or weeks occupying the pleura. And further, you may have the signs above described without the presence of pleurisy. Sharp pains of a nervous character not unfrequently closely imitate that of pleurisy, especially in hysterical females; and if they happen to be attended with feverish excitement, the resemblance is perfect. In fact, the greater number of the symptoms which are supposed to be distinctive of pleurisy, depend on a much exalted sensibility of the pleura, which is by no means a necessary accompaniment of its inflammation; and the symptoms of oppressed breathing, proceeding from the pressure of the effusion, may be distinct only when this effusion has accumulated very rapidly.

On the other hand, the physical signs in the greater number of cases are very unequivocal; and although they by no means speak of the degree or of the intensity of the inflammation, they seldom fail to announce its presence, and thus pretty accurately measure its most serious concomitant, the liquid effusion. I will now enumerate the signs of pleurisy in the order in which they commonly occur in the affected side, and we shall then consider the nature and value of each.

1. Diminished motion and sound of respiration from pain.
2. Sound of friction accompanying the motions of respiration.
3. Dulness on percussion in the most dependent parts of the chest, from the effusion.

4. Diminished motion, and sound of respiration, from the same cause.

5. *Ægophony*.

6. Cessation of vocal vibration felt by the hand.

7. Cessation of *ægophony* and all sound of the voice.

8. Enlargement of the side.

9. Displacement of the heart, liver, mediastinum, intercostal spaces.

10. Increased motions and sound of respiration on the sound side.

1. I believe that I formerly mentioned that the respiratory movements are so far within the control of the will, that they may be restrained in parts affected with pain ; and it is obvious that the sound of respiration will be diminished in proportion. This has been noticed by M. Andral as an early sign of pleurisy ; but you can readily perceive that it is a very equivocal one, since it depends merely on the presence of pain, which, as I have just remarked, may exist quite independently of inflammation. I may mention here, that when you examine the chest of a person who is suffering from pain, or who is shivering under the influence of cold or of the rigor of a fever, you will often be perplexed by the rumbling sounds produced by the slight convulsive action of the muscles of the chest, particularly of the back, which are sometimes enough to mask the respiratory murmur. You may distinguish them by observing that they are not stopped, but increased by the effort of holding the breath.

2. At the first onset of pleurisy there is sometimes heard a rubbing or creaking sound accompanying the movements of the chest. This may be owing to a certain degree of roughness or defective lubrication of the pulmonary and costal pleuræ at certain points, and when combined with the general symptoms may be considered a pretty exact sign, but it is very transient, and is not often heard. It may be produced, also, by interlobular emphysema, where it lasts for a much longer time. I am inclined to think, from a recollection of the cases in which I have heard this sound, that its production is favoured by the lung being partially distended or pushed against the walls of the chest during their motions ; for it is especially apt to occur where the lung is displaced by a tumor, or by a pleuritic effusion confined by adhesions ; and where the lung is partially distended with tuberculous or other deposits. In any of these cases you can understand how a portion of lung, being pressed against the ribs as they rise, may occasion a sound of friction ; and you may imitate it in the dead body by rubbing a piece of distended lung on the interior of the chest. The friction sound of pleurisy is commonly heard about the middle parts of the chest ; it generally ceases as soon as the sound of percussion announces the accumulation of liquid ; but in dry pleurisy, and in the cases of partial pressure, just alluded to, it may continue for a long time.

3. In by far the greater number of cases of pleurisy there is an effusion of serum soon after the commencement of the inflammation,

and the accumulation of this liquid in the chest is the cause of the signs by which pleurisy can be best distinguished. This fluid will accumulate first in the lowest parts of the chest, floating, to a certain extent, the lung upon it. Hence these parts will sound more or less dull on percussion, whilst the higher parts retain their usual resonance; and to some degree, change of posture, by changing the place of the liquid, will alter the situation of these sounds. As, however, the external vesicular structure yields more readily to pressure than the tubular parts within, the accumulating fluid soon mounts up in the form of a thin layer, between the lung and the ribs, to a considerable height in the chest. This thin layer slightly impairs the sound on percussion; and this more distinctly if the percussion be gentle and abrupt, as by flapping on a finger tightly applied, and comparing the sound, as usual, to that of corresponding parts of the opposite side. This sign as well as those to be next described, are liable to modifications from adhesions previously existing between the pulmonary and costal pleuræ. These we shall notice afterwards.

4. This same accumulation of liquid must, as you can easily perceive, diminish the extent of the motions of respiration in proportion to its bulk, which has taken the place of the most expansible part of the lung. The sound of respiration will, for the same reason, be weakened, and its duration shortened on the affected side. These signs need no further explanation.

5. About the same time at which the dulness on percussion and diminution of the respiratory murmur reach the middle regions of the chest, the vocal resonance there presents a remarkable modification. It is heard much more distinctly than is usual in those regions and it is superficial, as if produced in the spot, separately from the oral voice, and changed to a sort of small bleating trembling note, which so much resembles the voice of a goat, that Laennec has well termed it *ægophony*. This modification of the voice is heard most distinctly between the third and sixth ribs, which corresponds with the situation of the middle size bronchial tubes; but about the spine it is generally mixed with a louder and more uniform resonance, more of a common bronchophony, from the larger tubes at the root of the lung. Now how is it that a layer of liquid, which pushes the lung aside, can make the voice more audible than usual! Why does not the liquid condense the porous tissue of the lung, and thus make it a better conductor of sound? Then you will understand something of the modified character of the voice, if you consider the nature of the matter which it has to pass through, a thin layer of liquid, which, being thrown into active vibrations by it, trembles and dances in an irregular manner, now checking the sound, now transmitting it with increased force, so that the voice comes through tremulous and wiry. The high tones of the voice are those which are best transmitted in this way; for as I mentioned to you formerly, the bass tones do not enter the small tubes, but if strong, pervade the whole tissue with a diffused fremitus. Hence you will

hear ægophony best in women and children, and those who have high voices. In persons with a bass voice it is more commonly limited to the inferior angle of the scapula, or near the spine, and from its being seated in larger tubes, takes more the character of bronchophony ; but even there, if the layer of liquid reach to that height it will have something of a buzzing and bleating note. As the liquid increases, the ægophony becomes weaker, more distant, and loses much of its flutter or tremor, having now rather the sound of a diminutive deep seated voice, or a silvery echo of the original. This is because the lung is pushed so far away from the walls of the chest, and its tubes so much compressed ; and as these circumstances increase, the sound ceases altogether. It is not easy to determine what quantity of effusion is enough to do this ; but I am inclined to think that much sound of the voice is not transmitted when the layer of serum exceeds an inch in thickness. If the ægophony continue stationary for several days, it is a proof that the effusion is moderate, and does not increase rapidly, which is a favourable sign ; but it is often very transient, and many cases of pleuritic effusion are discovered after they have passed the degree which causes ægophony. Old adhesions will, however, modify this as well as the other physical signs. When ægophony is most distinct, it is often coupled with bronchial respiration, especially between the scapulæ, where also there is a good deal of common bronchophony with it. M. Renaud, a very good auscultator and able pathologist, has lately confirmed the opinion of Laennec, that ægophony is a kind of bronchial voice modified by its transmission through a layer of liquid. He observed in a pleuritic case, that the ægophony heard at the lower angle of the scapula when the patient was sitting, became changed to simple and louder bronchophony when the patient lay prostrate or stooped much forward ; this change of posture having the effect of permitting the liquid to gravitate to the anterior part of the chest, and floating the lung into contact with the parietes. Ægophony and bronchophony are different enough when their characters are well marked ; but they often present mixed and doubtful varieties that do not admit of such easy distinction. You must be practised in listening to both, before you can easily distinguish them ; but as far as description goes, I would represent the true character of ægophony to be a certain *tremulousness* in the voice when it is superficial, and an echo-like slenderness when it is deep-seated ; whilst bronchophony may present many other varieties.

6. An early and very characteristic effect of the accumulation of liquid in the pleural sac, is its intercepting the diffused vibration of the voice, which is usually felt by the hand applied to the chest. I formerly explained to you that this vibration is caused by the voice pervading the common tissue of the lung, and you can readily conceive that a layer of liquid would muffle and destroy it ; and it does this even when ægophony may be heard at the same spot, the vibrations of the latter being of too fine a kind to be felt by the hand. You have here also a distinction between a liquid effusion

and a solidification of the lung ; for the latter transmits the vocal vibrations with unusual force from the tubes. This diagnostic sign we owe to M. Reynaud ; and it is the more valuable because it is so easily obtained, even by a person who does not practise auscultation. It must not, however, be always considered as quite conclusive ; for there are some exceptions to it, both positive and negative. For example, in case of partial adhesions of the lung to the chest, there may be even more vibration than usual felt at the adhering parts where the lung and its tubes are pressed into close contact with the walls of the chest ; and it may happen, on the other hand, in solidification of the lung, that liquid or other obstruction in the bronchial tubes may prevent the voice from being transmitted through them.

7. As the liquid effusion increases, the ægophony and all sound of the voice ceases throughout the affected side, except within two or three inches of the spine, and in spots where the lung may have been adherent, which frequently happens at the upper part of the chest. The sound of respiration is also abolished in most parts of the chest, but never in the interscapular region, and rarely under the clavicle ; it is, however, much weaker in these parts than on the sound side, and may probably be only transmitted from that side. I have met with several instances in which the respiration had been audible to the last under the clavicle, yet on examination after death the lung was found entirely compressed against the spine. A word more presently about the influence of adhesions.

8. Enlargement of the affected side is the next of the signs of pleurisy that I have named. As you may suppose, the effusion must be pretty copious to render this enlargement perceptible ; but a difference between the two sides may sometimes be seen on inspection of the chest in different periods of respiration, where the quantity of liquid is not very great. The affected side is first seen to be larger at the end of expiration, when it does not diminish equally with the other side, especially in its lower portions. So if you encircle the chest with a piece of tape, fixing it at the sternum and at the spine, you will see it tighten and slacken with inspiration and expiration more obviously on the sound than on the diseased side, which remains more fixed in a state of partial distension. As the effusion increases, the difference is perceptible during the whole respiratory act, and the eye can easily detect the want of symmetry, whether the inspection be made in front, behind, or, as I formerly described to you, from above, looking downwards on the patient's shoulders. To be more exact, however, you must measure the chest, by encircling it this way horizontally with a piece of tape or ribbon, making it meet at the middle of the lower end of the sternum ; then taking the tape by the point where it crosses the spinous processes of the vertebral column, thus,—you have at once the measure of two sides to compare together. By the way, you see a considerable discrepancy between the two sides of this statue ; it is, nevertheless, an accurate cast of an antique marble ; and this shows

that the sculpture of the ancients is not always so perfect in accuracy and symmetry as it is said to be. Now in comparing the measurement of the two sides, you must bear in mind that the right side is, in the healthy state, from a quarter to half an inch larger than the left; and you must not trust to slight differences in favour of the right. The enlargement of the side is sometimes discoverable by the eye, as well as by measurement, two or three days after the first attack of the pleurisy; and it does not generally proceed afterwards in proportion to the effusion, until this becomes excessive, and has displaced the adjoining parts to a great extent.

9. Then we have a very important class of signs arising from the displacement of certain of the walls and organs bounding the effusion. Laennec had remarked that the intercostal spaces on the side of the effusion do not present their usual depressions, and are sometimes, especially in chronic cases, even prominent beyond the surface of the ribs. This is scarcely perceptible, however, in acute pleurisy, unless the subject be thin. In such cases I have seen the intercostal spaces not only prominent, but presenting also an evident fluctuation. In looking for this sign you should place the patient obliquely with regard to the light; and you may sometimes perceive it more readily by surveying him from a little distance, as from the foot of his bed, than by a closer inspection.

But we may generally learn more from the displacement of the organs adjoining the effusion, especially that of the heart and of the liver. These displacements had been noticed in empyema by surgical writers; but Laennec does not seem to have paid attention to them, and I believe that we owe to Drs. Stokes and Townsend their proper application to the diagnosis of liquid effusions in the chest. The displacement of the heart by an effusion in the left pleura is the most valuable and easily recognised of these. In this case the pulsations of the heart are felt and heard most distinctly under, or to the right, of the sternum, or in the epigastrium, instead of, as usual, between the cartilages of the fourth and sixth left ribs. Again, when the effusion is on the right side, you find the liver pushed down below the margins of the ribs, and you can trace its position both by feeling and by its dull sound on percussion. It is sometimes depressed so much as to form a sensible tumor in the abdomen; and I have known more than one case of latent pleurisy, in which this tumour was long supposed to be the chief disease, and the case was treated accordingly. The effusion in the right side, when considerable, also displaces the heart further than usual to the left, and you may sometimes feel it pulsating to the left of the mamilla, and even below the axilla. Dr. Stokes has lately published some interesting observations, with the view to prove that the displacement of the diaphragm and intercostal muscles is much favoured by a paralysis of their muscular fibres, the result of inflammation in the serous membrane covering them.

The displacement of the mediastinum is to be discovered only by percussion. Situated as this is naturally, in the mesial plane, it

divides the two cavities of the pleura at a line down the middle of the sternum, which bone sounds well on percussion from the margins of both lungs, which lie under it. But a copious effusion will push the mediastinum towards the opposite side, and, by occupying the space behind the sternum, will give this bone a dull sound on percussion; and this may extend even half an inch beyond it. This dulness is most perceptible below the juncture of the second rib, for above this the resonance is seldom destroyed, and often has an amphoric character from the great air-tubes, which are not compressed. This is still more remarkable in some cases of adhesion which I shall notice presently.

Now all these displacements may also be produced by an accumulation of air in the cavities of the chest; but you can perceive at once that percussion would give a prompt distinction between these two cases, by the tympanitic sound in case of air, and the dulness in case of liquid.

10. In all cases of physical examination, you must avail yourselves of the standard of comparison which the two sides offer, and you will often find that the sound side will give you not only the negative proofs of the absence of disease in it, which may well be compared with the positive signs of disease on the opposite side, but it will even show you an exaggeration of the signs of healthy action, in consequence of its work being really increased. Thus you will see the healthy side move more fully and quickly than usual, and the sound of respiration will be increased there in a remarkable degree, so as to resemble the loud respiration of children; wherefore it is called *puerile*.

I might enter into many other particulars respecting these different signs, but we have not time to do so; and, indeed, if we had, I would rather leave for your own observation and reflection the less essential details which present themselves in practice. These diagrams, which represent by sections of the chest the position of different quantities of effusion, as I have found them after death, and their effect on the chest and its organs, may serve to impress the physical signs more strongly on your memory. In fig. 1, you see indicated by the shading a moderate effusion on the right side, and the manner in which it displaces a lung unrestrained by adhesions; ægophony is more or less heard from *a* to *b*, and the sound of percussion is impaired, but not dull; but below *b* it is perfectly dull. Fig. 2, represents an abundant effusion on the left side, which compresses the lung against the mediastinum and spine, displaces the mediastinum and heart to the right of the sternum, gives a prominence to the intercostal spaces, and depresses the diaphragm. You would suppose that such a quantity of liquid would render the sound of percussion quite dull on the whole of that side; but it commonly happens that percussion above *a*, gives a sound which is not perfectly dull, for it receives a slight degree of resonance from the opposite lung; below that point the heart cuts off that source of resonance, and the sound is quite dull.

Fig. 1.

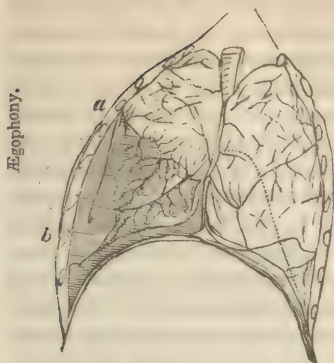
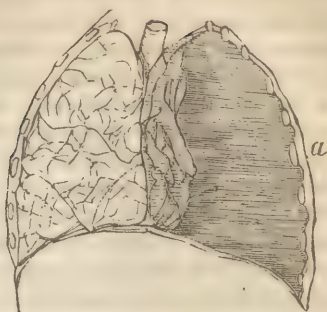


Fig. 2.



I have before stated to you that the physical signs of pleurisy are liable to be much modified by old adhesions, which bind the lung to the walls of the chest; and some of these modifications are so singular and important in relation to diagnosis, that I must advert to them. When the adhesions are loose, they only form bands or cells distended with fluid; and, keeping the lung at a moderate distance from the walls of the chest, they may render the continuance of ægophony much longer than it would be without them. When an adhesion is so close and strong that the accumulating fluid cannot separate it, the lung is then compressed against it; or if there are several adhering points, the attachments to these are preserved by so many pillars of compressed lung. You see this represented in fig. 3, where the compressed lung still adheres to the upper part of the chest, and also to the diaphragm and mediastinum; and mark the effect: instead of a total abolition of the voice and respiration, you have at the part corresponding with the upper adhesion a loud bronchophony and bronchial respiration transmitted

Fig. 3.

Bronchophony.

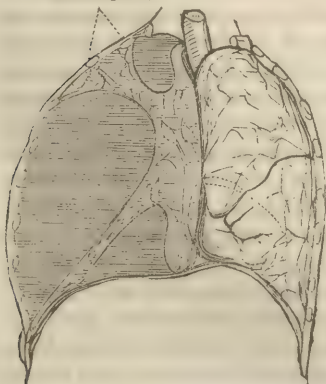
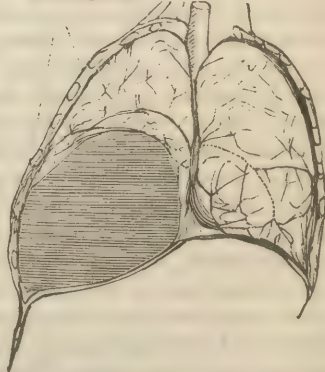


Fig. 4.

Bronchophony.



from the large tubes by the adhering dense column of lung ; and the same thing not uncommonly occurs in other of the upper and middle regions of the chest. Fig. 4, represents a pretty common form of partial effusion. The upper lobes of the lungs are the most liable to close adhesions ; and when thus adhering in consequence of former disease, the lung is compressed by the effusion below against the whole upper part of the chest. In this condition it may still admit air ; but as its vesicular structure is much compressed, the sound of respiration will be tubular or bronchial, and a noisy bronchophony will be transmitted by it to the whole upper region of that side. I have often heard the voice and respiration quite tracheal from this cause ; and I have been more than once deceived by it into the belief that there were caverns underneath. The displacement of the heart and liver, the prominence of the intercostal spaces, and the dulness on percussion of the whole lower portion of the sternum, together with the enlargement of the affected side, will generally distinguish the true nature of these cases. Fig. 5, repre-

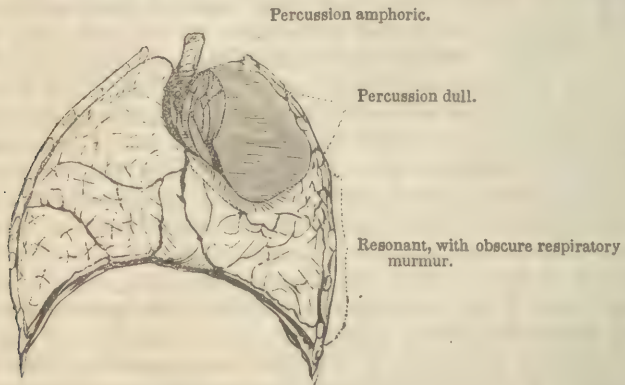


Fig. 5.

sents a much more rare kind of partial pleurisy ; but I have met with it in some remarkable instances, and the phenomena were so extraordinary in two of these, that I think it necessary to mention them, lest you should in any case be misled by them, as I was in the first instance. In both these cases there was moderate sound on percussion below the fourth left rib, and the respiration could be heard there obscurely ; in one case it was bronchial. The heart was felt to pulsate in the epigastrium, and to the right of the sternum. From about the second to the fourth rib the sound on percussion was dull ; but above this the sound was just of this bottle-like character, that you hear when I fillip on my finger applied to the wind-pipe. It is a kind of tympanitic sound, and as in one of these cases there was also a sort of amphoric respiration heard in this spot, I concluded the case to be one of pneumo-thorax from perforation of the lung. This patient, whom I saw with my friend

Dr. Roscoe, surprised me by soon getting well, and loosing all those signs, which made me reflect more on the matter; and having since met with a similar case which proved fatal, I have satisfied myself as to the cause of the phenomenon. Dr. Hudson, of Dublin, has also recently described cases in which a loud tympanitic sound on percussion was presented in the upper part of the chest of a patient affected with pneumonia. Now you will understand how this sound is produced if you will listen to this tracheal sound which I get by filliping on my windpipe above the sternum. The windpipe also lies under the sternum, and it divides into the two great bronchi, which spread between one and two inches below the clavicles. Here, however, the porous lung lies over these tubes, and intercepts their resonance on percussion; but let this portion of lung be *perfectly* condensed by a liquid effusion, or perfectly consolidated by hepatization, and you will then get the bottle-note of the tubes, just as you do of the wind-pipe where no lung intervenes. The reason why this phenomenon does not occur more frequently is, that it does not often happen that the compression or solidification of the upper lobe is complete enough; but since my attention has been drawn to it, I have met with several cases of both pleurisy and pneumonia, in which it existed in a smaller degree; and I had occasion to notice in the last lecture that it sometimes occurs with dilated bronchi.

LECTURE XVII.

Diseases of the Pleura (continued)—Further Effects of Pleurisy: Solid Effusions; their Effects: Contraction of the Chest; Signs and Effects: Liquid Effusions; Empyema—Relations of Acute and Chronic Pleurisy—Treatment of Pleurisy; Acute; Chronic—Paracentesis Thoracis; Indications and Objects of the Operation; Causes of Failure—Directions for the Operation.

IN the last lecture we were occupied with the pathology and signs of acute pleurisy, from its commencement to its period of fullest effusion. Before we can understand the further history of pleurisy and of its consequences, we must revert to the pathological changes which accompany it.

If, in consequence of the treatment, or of the mildness of the irritation and its relief by the effusion, the inflammatory orgasm has ceased, the re-absorption of the fluid and lymph is a matter of course, and the ordinary efforts of respiration may suffice to expand the lung in proportion as this absorption proceeds. You will then have a gradual diminution and removal of the signs which marked the increase of the effusion. I need not go over these in detail, for if you know how the signs are produced, you will as readily perceive how they are removed. The return of ægophony, and of the sound of respiration, which reappears at the parts where it was last heard,

generally the upper parts of the chest, are the most available signs of improvement ; and these, together with diminution of the side, and a gradual improvement of the sound on percussion, are to be watched from time to time as proofs that the improvement is progressive.

In very moderate cases, the liquid is absorbed away before the lymph or albuminous matter is removed ; and when the pleural surfaces covered with this come together, a sound of rustling or rubbing is sometimes heard, which soon ends in the permanent adhesion of these surfaces by bands of false membranes. Now if these false membranes are formed after the liquid has been removed, and the lung has recovered its full extent of expansibility, they are adapted to its free motions, and do not to any material extent interfere with them. Hence, we often find in dead bodies adhesions which are lengthened in the lower parts of the chest, where, from the action of the diaphragm, the lungs descend as the ribs rise, whilst in the upper parts the adhesions are short, because the lungs there follow more exactly the movements of the walls of the chest. But in severer cases, which are also very common, inflammation continues after the liquid has been abundantly poured out, and not only increases and perpetuates this liquid effusion, but throws out albuminous matter also in various conditions, which, by its present qualities or future changes, may produce a variety of prejudicial effects, all tending more or less to interfere with the perfect restoration of the organs to a healthy state. Now, arising, as these consequences do, out of continued inflammation, which either has been imperfectly treated, or has been neglected from being latent, they are strong proofs of the value of the physical signs, which are never absent, and which will scarcely ever fail to declare the presence of the lurking mischief. Let us now glance at some of these solid products of pleuritis.

1. Healthy and highly organizable lymph, when deposited in a thick layer, must in some measure restrain the expansion of the lung, and thereby retard the absorption of the fluid. This lymph may be diminished by absorption ; and the membranes formed of it may ultimately adapt themselves to the full expansion of the lung ; but there will be less chance of this, in proportion as the liquid effused is copious, and its removal slow.

2. In cases similar to that just mentioned, but with a lymph less organizable, the product of a less active inflammation, or in which there is much of the colouring matter of the blood, its organization is more tardy, and the membrane resulting is more rigid, and of a less yielding nature ; consequently the lung is more permanently confined in its compressed state. The membranes which are formed on the pleura in these cases are sometimes quite cartilaginous in density, and of considerable thickness ; and I have met with several examples in which they have become partially ossified. If these are formed, and acquire their density before the liquid has been removed, it is quite clear that they must for ever bind down the lung ; but I have seen several cases in which there have been proofs of

contraction after the absorption of the liquid ; and I should explain this by the fact that certain newly-formed tissues have a tendency to contract for some time after their production. You know how remarkably this is exemplified in the contraction of newly-formed skin after burns and other extensive wounds of the surface. It is also shown in the puckering of the tissue around false membranes lining cavities in the lung, in the contraction of the valves of the heart, and in many other cases where false membranes have been slowly formed, and tend to assume a fibrous or fibro-cartilaginous, rather than a serous or cellular, character.

3. But the inflamed pleura may throw out a lymph of still lower vitality, susceptible of but imperfect organization, and wholly incapable of throwing out more of an organizable character ; hence, when the pleura is coated with it, if the inflammation continue, the overflow of the nutritive secretion will be in the form of a curdy matter, or of mere loose shreds of albumen.

4. The solid matter may be thrown out in a disintegrated state, utterly insusceptible of organization, and diffused through the fluid in flakes or particles, forming a mixture more or less resembling pus, which is the fluid of empyema. Although in many instances this is the result of a less active form of pleurisy, and owes its persistence or tendency to increase to the want of vitality in its solid matter, yet we do meet with cases of empyema which arise from very acute forms of inflammation. In these instances the fluid is more strictly purulent, the solid matter being in form of globules like those of pus, and seems to be the result of what may be called a suppurating diathesis, in consequence of which all the albuminous products of inflammation tend to assume a purulent character ; nay, in such a case, I have seen pus formed within fibrinous clots of the blood itself in the heart ; and it is difficult to avoid the supposition that it is something in the condition of the blood that determines this less usual product from the acute inflammation of a serous membrane.

5. Lastly, as the solid accretions on the inflamed pleura, you may have the various morbid productions called tuberculous, scirrhus, fungoid, and melanotic ; these being commonly the result of some constitutional taint developed by the local inflammation, but sometimes also, as I believe, the product of peculiar modifications of the inflammation itself. This, however, is a subject that we cannot enter into at present.

Now you are not to suppose that the products of pleurisy, in every case, belong exclusively to one of the kinds now described, or that they are so simple as to be rigidly divisible by such a classification. You will rarely examine a case of pleurisy, after death, without finding traces of several forms of the products of inflammation : you cannot examine many without perceiving that these pass by insensible gradations into one another, and that although you may meet with many that are clearly referable to one denomination, you will find others in which the products of inflammation are mixed or intermediate. I assure you that I have long sought for a positive line of physical demarcation between lymph and pus, pus and yellow

tubercles—between false membranes, serous, fibrous, and cartilaginous ; and I have sought in vain ; for although, in a majority of instances, it has not been difficult to say which name is most applicable, yet cases not unfrequently present themselves in which these products of inflammation have such a dubious and intermediate character, that it is difficult to avoid the conclusion that they pass by insensible gradations into each other. This is not a matter of merely speculative interest ; for if we find proofs of this transition in different instances, there is full reason to suppose that it may take place in the same case ; and it becomes a matter of great importance to determine under what circumstances such transitions take place, and how they may be influenced so as to be directed in the most favourable way.

If you reflect on the consequences of all these products of prolonged inflammation of the pleura, you may perceive that they all tend to keep down the lung in the compressed state to which it was reduced by the first effusion, and they do this by the rigid false membranes which are formed when the solid effusion is susceptible of organization, and by the persistence of the liquid effusion when the solid matter is destitute of organization, and acts as an extraneous irritant. We must suppose, too, that the absorbing properties of the pleura must be altered by the long continuance of disease, and that various lesions are propagated to the adjoining tissues ; which, however they may have escaped the immediate effects of inflammation in its acute form, can scarcely fail to be affected by the changes of nutrition and action induced by the slow, but less limited influence of chronic inflammation. Hence the parenchyma of the lung, the bronchi, the pericardium, the bones and cartilages of the chest, occasionally become the seat of various changes. Thus the lung becomes at first consolidated and then atrophied ; the tubes secrete pus and become dilated ; the pericardium forms adhesions to the heart, and becomes thickened ; and in cases of empyema, the ribs, vertebræ, and their ligaments, sometimes become carious. Nor must we forget the unfavourable operation of the disease on other functions ; the obstruction to the circulation by pressure on the pulmonary and adjoining vessels, the abridgment of the function of the lungs themselves, and the irritating or depressing influence occasionally extended from the seat of lesions to the various organs of the abdomen. In fact, besides the injury done to the respiratory organs, an imperfectly cured pleurisy may, in an insidious manner, oppress the whole system, and bring it into an anomalous cachectic state, in which morbid conditions of various kinds may be produced or brought into activity. Did our time admit, I would illustrate by many cases all these effects and sequels of pleurisy ; and these cases would serve to show, too, that the form of disease that leads to such results, is often of the most insidious kind, either marked by no prominent symptoms at all, or losing them before the mischief is half subdued. But we have not time for these illustrations, which belong rather to a course of clinical medicine ; but your own observation in hospitals or private practice will soon supply you with them, for such cases are by no means uncommon.

Now, for practical purposes, let us divide all these cases into two classes :—1st, those in which absorption ultimately predominates over effusion, and the liquid is sooner or later removed ; and 2dly, those in which effusion predominates, and the liquid can only be removed through a perforation of the pleura. In the first of these, as the absorption of the fluid proceeds, what is it that supplies its place ? The lung, as we have seen, is either so bound down by rigid false membranes, or so condensed and obliterated by the long continued pressure, that it is not susceptible of its former expansion to effect this purpose. In the great majority of cases, as the liquid is absorbed away, the walls of the chest are contracted and fall in ; so that the diseased side, which at the height of the effusion measured, perhaps, an inch or two more than the healthy side, now gradually becomes considerably smaller, sometimes to the extent of two or three inches. The contraction is first perceptible in the upper part of the chest, and, with the depression and more fixed condition of the shoulder, contrasts remarkably, on inspection, with the full development and active motions of the sound side. There is still some apparent fulness of the lower part of the diseased side, and this appearance will often continue even when it measures less than the sound side. If you examine the contracted side more narrowly, you will see in detail that the ribs are lower at the sides and closer together, the scapula more prominent and nearer to the spine, and sometimes the sternum, and occasionally even the spinal column also, is curved concave towards this side. In all this you see the results of the atmospheric pressure, together with unantagonized muscular efforts, acting on the walls of the chest. You may also have atmospheric pressure, contributing to the same end, from the abdomen : thus the diaphragm is pressed permanently upwards, carrying with it the mass of the liver on the right side, and the resonant stomach on the left ; and on watching the abdomen, you do not see it swell on that side, as on the other, with the acts of inspiration. In some cases you will find that, even within the chest, the same atmospheric pressure is exerted from the sound side, causing displacements the very reverse of those which had been occasioned by the pressure of the previous effusion. Thus I have seen several cases in which the healthy lung had displaced the mediastinum towards the contracted side, not only under the whole sternum, but even under the cartilages of the ribs, to the extent of an inch beyond it ; so that these parts sounded well on percussion, and the intercostal spaces there showed the movements of respiration, which scarcely affected any other part of that side. Nay, in one instance, I have seen the heart drawn over to the right side, after a latent pleurisy affecting that side, so that it was felt pulsating to the right of the sternum.*

* Dr. Stokes, in his late work, has also described this mode of displacement of the heart. Soon after these lectures were delivered, I met with another case of the same kind ; the patient is now under the care of Dr. Symonds, of Bristol, who has lately informed me that the pulsations of the heart are still most distinct to the right of the sternum.

But I have not described to you the auscultatory signs of a side, contracted after pleurisy. In many cases, where the effusion has been copious and of long standing, the sounds of respiration and percussion continue imperfect permanently, although the liquid may have been completely removed ; and in almost every case they are more or less impaired for months after the attack ; in fact, they correspond pretty well with the appearance and diminished motion of the affected side, and are to be referred to the same causes. The improvement is generally to be found in the upper parts of the chest, and near the spine. With the return of a weak respiratory murmur, and slight resonance on percussion, some degree of vocal resonance may also accompany the removal of the liquid—in the superior parts of the chest, amounting to bronchophony—in others, being only the diffused pectoral fremitus, according to the size of the bronchial tubes, and the degree and permanency of their compression. The bronchophony in these cases is generally of a cracked or buzzing character ; and I have known it very loud and diffused over some extent of surface. This is one of those instances in which some physical signs may deceive you, unless you pay attention to others, and to the general history of the case ; for if, for the first time, you see a patient in this condition, and if he happen to have an attack of bronchitis, you may be led to believe that the resonance of the voice and dulness on percussion are caused by consolidation, by recent inflammation of the lung, or by tubercles ; but this error may be avoided by attending to the history of the case, and the signs of contraction that are also present. The dulness on percussion in the contracted chest is less owing to the absence of air, than to the compressed, drawn in, condition of the walls, which are no longer free to vibrate as usual ; and although there be air in considerable quantity in the lung underneath, this air gives no spring to counterbalance the atmospheric pressure, which is continually acting as a dead weight on the contracted side. Sometimes you may get more sound by pressing the fingers strongly on the side, and then striking on them ; this pressure brings the walls beyond the atmospheric weight, and within the influence of the contained air, so that they may then vibrate more freely. This is according to the principles of the sound on percussion which I formerly explained to you, and need not dwell on further.

You will observe that, in these cases of pleurisy, the condition of the side of the chest was, at the period of copious effusion, the reverse of what it becomes when that effusion is removed ; then it was dilated, and the adjoining parts pushed from it ; now it is contracted, and the adjoining parts are drawn into it. Is there not, then, an intermediate stage in which neither of these conditions is presented, and the side has the usual shape and dimensions ? My experience leads me to say no ; for the transition from one condition to the other is not generally uniform, but partial. The common case is, that the contraction begins in the upper part of the chest, before the dilatation and displacements have ceased in the lower ; and it

seldom happens that there is not, during the diminution of a pleuritic effusion, an irregularity in the shape of the chest, a comparative bulging of the lower portion, which may serve to distinguish it from the case of a consolidated lung.

In the cases which we have hitherto considered, the effusion was supposed to be general into the whole cavity of one side of the chest, and so was the contraction which succeeded it ; but what contraction can obliterate a cavity left by partial effusions, limited by rigid adhesions, such as you see in these drawings ? [See figures 4 and 5 in the last lecture.] I have had occasion to see the *post-mortem* examination of a few such cases : in two of them the cavity was not obliterated, but contained air, secreted, I presume, from the membranes, for there was no perforation of the pleura. In another there had been partial contraction ; but a considerable space remained, and was filled with a semi-solid curdy matter, like what is found in old vomicae, and probably of the same kind ;—but more of this by and by.

Well now, what would you think of these cases of contracted chest, whose respiratory organs are reduced by nearly one-half ? You would suppose them to be a crippled set of beings, capable of dragging on but an imperfect state of existence ; yet it is curious enough that there have been some such individuals who have continued to enjoy good health, and to be actively engaged in the pursuits of life. Laennec tells us of the case of a distinguished surgeon of Paris, who although he had one side contracted in a very marked degree, from an attack of pleurisy in his youth, yet enjoyed excellent health, and was in the habit of lecturing twice a day without inconvenience. I have known two or three instances of the same kind ; but I must say that here the contraction was not excessive, and the respiratory murmur was by no means abolished. In other cases—and they are, I think the most common—extensive contraction of the chest causes such an habitual shortness of breath, as to incapacitate the person from all active exertion, and to make any slight bronchitic attacks, or febrile excitement, cause severe and distressing dyspnoea. I have further had occasion to observe, that before the system becomes accommodated to the abridgment of respiration which this lesion produces, there is an enfeebled or cachectic state of the body, in which various trains of disorder may arise ; and unless care be taken to counteract them by remedies and circumstances most favourable to the general health, scrofulous or dropsical disorders may be engendered, and develop new mischief in the respiratory organs, or elsewhere. Although, therefore, we may look on contraction of the chest as a mode of curative termination of pleurisy, it is one of the least favourable kind, and liable to many detaching circumstances.

We have now to advert to the other class of cases, in which effusion preponderates over absorption, and the liquid can only be removed through a perforation of the pleura, this character is to be traced, in some instances, to the condition of the membrane, which either from its continued irritation, or from a change of structure,

secretes more than it can absorb. Sometimes the accumulative tendency of the effusion may arise from some obstruction in the circulation, dependent on disease of the heart or great vessels, or even on the partial pressure of the effusion itself. But the more common cause of increasing effusion is the nature of the matter effused, which, when of a purulent character is not readily absorbed, and constitutes the *empyema* of authors. This, when accumulating rapidly, proves a source of oppression to the breathing, and of immediate danger ; if slowly, of irritation, obstruction, and consequent disorder to the vital powers generally. With the presence of pus there is not uncommonly associated an ulcerative process, which may give vent to the matter through the lungs, the walls of the chest, or the diaphragm, and which in cases of long continuance, not unfrequently extends to the vertebræ and ribs, and their cartilages and ligaments, and to form extensive abscesses between the muscles and integuments of the chest and abdomen. The prejudicial nature of such consequences of pleurisy are too obvious to need further comment from me.

You may have remarked that in this review of the pathology I have not given any separate notice of *chronic pleurisy* ; and my reason is, that the transition of the acute to the chronic is so indefinite, and the symptoms of the recent disease sometimes have so little of an acute character, whilst that of a long duration occasionally manifests so much greater an intensity of irritation, that the terms acute and chronic would be less applicable to pleuritic affections than to inflammations in most other organs. And we can see some reason for this, in the fact, that the pleura being a shut sac, is liable to have its acute inflammations made a chronic disease by the retention of its product ; and chronic pleurisy is perpetually liable to be excited into an acute state by the distending or irritating influence of the effusion. Still, differences of disease are very apparent in many cases, in the prevalence of high inflammatory fever in some, and in there being no fever, or one of a hectic kind, in others ; in the sthenic condition of the circulation in some and its depressed weak state in others, whatever may be the degrees of pain or nervous irritation accompanying them. These differences, although not easily included in a general view of the pathology, must not be disregarded in clinical observation, nor in the direction of the treatment.

I shall be very brief in speaking of the treatment of pleurisy, for you will find this pretty fully described by recent authors. The most desirable object is to destroy the inflammation at its onset, when the only physical sign may be the friction sound, and before the signs show that the effusion is considerable. For this purpose, the most effectual remedy is a full venesection, carried, if possible, to such an amount as to remove all pain on full inspiration ; or if there have been no pain until all hardness of the pulse ceases. This should be followed by free leeching or cupping the affected side. I think leeches are rather to be preferred, but they should not be

applied sparingly, and should be immediately followed by a large warm poultice covered with flannel. These depletory measures must be repeated if within a few hours the pain returns, or the pulse resumes its hardness. Of internal remedies, those are most useful, in the first instance, which will aid the bloodletting in producing an impression on the circulation, especially brisk purgatives containing mercury and antimony, which act fully on all the secretions. Calomel and James's powder, followed by an active salts and senna draught, generally answer best. Tartarized antimony is less effectual in this than in other inflammations of the chest : and I have seen it do harm by exciting vomiting ; but in doses short of that effect it may prove useful.

It commonly happens that such measures take off the edge of the disease without destroying it entirely, which must be a work of time ; and although the pain, dyspnœa, and cough, be much relieved, they are not removed, and the physical signs show that the effusion to a greater or less extent has taken place. Under these circumstances the proper means are those which tend to remove the remaining inflammation, and to promote the absorption of the matter already effused. The most powerful of these is mercury, which may be combined with enough of opium and ipecacuanha, to prevent it from passing off too freely by the bowels ; and to this may be added digitalis or colchicum, in a saline mixture with an excess of alkali, to keep down the action of the heart and arteries, to lower the inflammatory condition of the blood, and to determine to the kidneys. The beneficial influence of mercury is sometimes apparent when it does not affect the gums, especially in young subjects, its operation being only manifest on the hepatic and alvine secretion, which is green, dark, or high coloured, from different conditions of the bilious matter in it ; but in more cases the gums exhibit its operation before these excretions are produced.

Venesection can seldom be repeated with much advantage after the first few days, unless on the occasion of a fresh access of pain, or other symptoms which prove the renewal of acute inflammation. Occasional leechings continue to be useful ; but after the inflammatory fever has been reduced, the most effectual external remedies are blisters, which should be used large, and not left on too long ; from six to eight hours is generally time enough to make them vesicate without irritating the system by the absorption of their matter. Where the effusion is abundant, a succession of blisters will be necessary ; or they may be varied by a suppurating counter-irritant, such as the tartar-emetic ointment or solution. The diet, of course, must be of the most starving kind in the first instance, and the patient must remain as quiet as possible in bed. When the inflammation is subdued, on the other hand, sitting up, and if he can bear it well, using a little exercise about the room, will tend to promote the absorption of the fluid. If, from insufficiency, or in spite of treatment, the signs of effusion continue beyond two or three weeks little or not at all diminished, with more or less constitutional distur-

bance, it is to be apprehended that the disease may take a chronic form in which the character of treatment must, to a certain extent, be changed. If the strength continue to be lost, the pulse being weak, and the fever, if any, of a hectic kind, a more nutritious and tonic plan must be cautiously pursued to the extent that the patient can bear it; the heat of skin, pulse, cough, and condition of the breathing, being referred to as tests of the suitableness of the change. But external counter-irritation should still be continued, especially by blisters, which may, perhaps, be useful, not only to remove internal inflammation, but also to restore energy to the external muscles of respiration, as they do sometimes to those of a paralysed limb. The secretions must be kept free by medicines of a milder class than those used in the acute stage; and except with this view I have not found much benefit from the use of mercury in the late asthenic stages of simple pleurisy. In fact, when the effusion has a purulent character, I believe the constitutional action of mercury to be injurious. In these stages I have seen the most salutary effects from the employment of the hydriodate of potass, in the dose of two or three grains, three or four times a day; or in more asthenic cases, the iodide of iron, in rather smaller doses. To preserve the latter from decomposition, it should be kept in solution with a piece of clean iron or a coil of iron wire in the bottle, as recommended by Mr. Squire. As there is occasionally apt to be, in both these medicines, some free iodine, which often occasions gastric irritation and nervous symptoms, I always desire patients to eat a little bit of bread or biscuit after each dose; the starch in this combining with the free iodine, removes its injurious property. These medicines act on the kidneys, and seem especially to promote absorption; and I know of none which seem so often to succeed in hastening the removal of a pleuritic effusion. Various other medicines may be useful in particular cases; but we have no time even to name them. Where a dropsical diathesis prevails, the tartrate of iron is also sometimes a very good diuretic. Gentle exercise is serviceable in promoting the expansion of the chest and lungs, and the absorption of the fluid; and in the more chronic cases, it is of the utmost importance to promote the general health by free exposure to a pure, mild, and suitable air, which in cases disposed to scrofula, will be best found at the seaside.

It only remains for me now to say a few words on the subject of tapping the chest, an operation which, although belonging properly to the province of surgery, should only be performed under the guidance of a competent knowledge of the pathology and physical signs of pleurisy. There are two kinds of cases in which it becomes proper to give exit to the liquid accumulated in the pleural sac. One includes the examples of the recent disease in which the effusion takes place so quickly and abundantly as to endanger life by the pressure which it causes on the lungs. A sudden effusion may have this effect, where its quantity is not sufficient to compress the lung totally, or to displace the viscera to a great extent; but there

will be always some enlargement of the side ; which, with the dullness on percussion, and the absence of respiration and vocal resonance, will sufficiently indicate the nature of the case. Here the liquid is generally serum with more or less lymph, and it generally deposits a further clot of gelatinous fibrin after it has been drawn from the chest ; but the same liquid is sometimes yielded by pleurisies of long standing. The other class comprehends those cases in which the pleurisy has existed for a longer time, and the effusion, instead of showing a disposition to disperse under the influence of remedies, either increases or remains stationary, and whether it cause a dangerous degree of dyspnœa or not, it must cause mischief by perpetuating the compressed state of the lung, as well as by the various other structural and functional affections which I have before alluded to. The cases of true empyema are generally included in this number, and are considered the more legitimate subject for operation, because it is believed that there is little chance of purulent matter being removed by absorption ; but I must add, that experience proves that the operation is not so often successful in these cases as where the effusion is not purulent.

Now the object of the operation is to remove the fluid, which either from its quantity oppresses functions, or by its quality extends and perpetuates structural lesions within the chest. With this view an external vent is given to it, any number of times, until its quantity is so far diminished, and its quality improved, that it shall not prevent the re-expansion of the lung, as far as that is possible, and the contraction of the chest to fill up the remaining deficiency. But you can easily perceive that, after the first pressure is relieved, the remaining fluid cannot be drawn out of the chest without something to occupy its place ; and unless great precautions be taken, that something will be air, not drawn into the lung, but through the orifice into the cavity of the chest. Now air thus introduced often seems to have the effect of causing an unfavourable change in the remaining liquid, rendering that which was serous decidedly purulent, and giving to pus a fœtid character. Further, when air gets access, it tends to do mischief, whether the orifice remain open or be closed : in the former case, the air passing in and out prevents the lung from expanding, and constantly irritates the serous membranes, which are not fitted for contact with it ; and if it be closed, the air admitted tends to engender more air, by the decomposition which it causes in the remaining liquid, so that the pleura soon becomes as much distended as before the operation. Hence, although the operation generally gives temporary relief, it has often been followed by symptoms of irritation, or of increased oppression, which have ultimately led to an unfavourable result. The cause of failure in some cases, it is true, is irremediable disease in the walls or viscera of the chest, or perhaps in the constitution, which may be either the cause or the effect of a long continuance of the pleuritic effusion ; but even in these cases the operation may prove the means either of great temporary relief, and considerable

prolongation of life, or of just the contrary, according as it is performed or not with due reference to sound physiological and pathological principles. The operation has, it is true, sometimes succeeded where these have not been much attended to; but this has been for the most part in young subjects, where the reparatory powers are active, and sufficient to countervail very unfavourable circumstances; and I feel confident that it would succeed in many more instances, were it resorted to at the time and in the way pointed out by the structure of the parts, and their properties in health and disease. One great error has been to delay the operation too long, until some of the bad consequences of rigid membranes, obliteration of the tissue of the lung, surfaces secreting unhealthy matter, and incapable of adhesion, ulcerations and even sloughing of the soft parts, caries of the bones, morbid deposits in other parts, and depression of the vital powers generally, prevent the possibility of recovery. This error having arisen in some measure from some uncertainty in the diagnosis of liquid effusion, ought now to be prevented by the improvement of our knowledge in this respect. Another great error I consider to have been, a neglect of all means to promote the re-expansion of the lung, which is the only natural mode of supplying the place of the fluid drawn off, and is an obvious step towards a restoration of the healthy condition and function of the parts. The common notion is, that the fluid should be drawn off, without considering whether anything more hurtful may occupy its place.

I find that I have not time to describe to you the different modes of operation that are recommended by authors, or that I have seen performed, so I will merely tell you what appears to me to be that which promises the best chance of success, according to the views which I have been explaining to you. The spot for the introduction of the trocar must be determined with due reference to the physical signs, carefully avoiding every part where, or near which, there is any sound of respiration, resonance of the voice, or not perfect dulness on percussion. A projection and fluctuation of an intercostal space give a greater eligibility to a spot; and these circumstances present themselves most frequently at the inferior lateral parts of the chest, from the fourth to the eighth rib, where also the soft walls of the chest are as thin as any where. In all cases it is a proper precaution to pass a grooved needle first, as recommended by Dr. T. Davies, for this will determine the presence of the liquid, and in some degree its nature, and the thickness of the walls which contain it. Of course it is proper to avoid the immediate neighbourhood of the heart, or any of the known arteries or nerves. The upper margin of the sixth rib most commonly presents a favourable spot, but whether more or less at the side must be determined by the position of the heart and other circumstances. Now as I hold it to be the great object to not only remove fluid, but promote the restoration of the function of the lung, and therefore to prevent the admission of air by the puncture, I would have the whole operation

conducted with this view. The patient should be lying on his back, or not more propped up than is necessary for the state of his breathing. The puncture, instead of being in the most dependent part of the chest, should be made as near the front as is consistent with the safety of the viscera and other considerations just alluded to. The trocar should not be pushed in further than is necessary to clear the parietes ; but the canula may be pushed in further after the stillette is withdrawn, and its sides should have several holes in them. The skin should be drawn aside, so that the puncture through it may not, after the trocar is withdrawn, correspond with that in the costal pleura, but may form a valvular orifice. As soon as the stilette is withdrawn, steady pressure should be applied, by the hands of assistants, to the affected side to depress the ribs and shoulder, and to press up the diaphragm, to promote the flow of liquid, and to prevent the introduction of air through the orifice, during any sudden and forcible act of inspiration. For the same reason, during a fit of coughing, if there appear any tendency to intermission in the stream of liquid, the orifice should be closed by the finger. The pressure should be steadily increased as the liquid flows : and if the stream should stop suddenly, a probe should be passed through the canula to clear it of clots of lymph or other obstructing matter, and this may be done also if the stoppage is more gradual ; but if still no more flows, the canula should be quickly withdrawn, and a compress, or a large poultice, placed on the orifice ; and then, but not till then, the pressure on the walls of the chest may be withdrawn. What will be the result ? The walls of the chest, expanding by their own elasticity on the removal of the pressure, will draw air into the compressed lung, which being thus expanded will begin to resume its part in the function of respiration and circulation, and will thus promote the absorption of the rest of the fluid, and improve the condition of the whole system. Even if the fluid should accumulate again the temporary expansion of the lung will have served to restore its condition, so that when another quantity of fluid is again withdrawn, its tissue will be the better prepared for a restoration of its function. When the fluid is of a decidedly purulent character, or if otherwise its absorption is difficult, it may be useful to displace it, by injecting through a double-tubed canula warm water ; to which may be added, in some cases, a weak solution of nitrate of silver or some other medicament, which may improve the condition of the membrane, and dispose it to secrete adhesive lymph instead of pus.

LECTURE XVIII.

Diseases of the Pleura (concluded)—Hydrothorax : its Causes—Signs and Treatment—Pneumothorax—From Adhesions—Spontaneous—From perforation of the Pleura—Signs and Symptoms—Metallic Tinkling, &c.—Treatment—Altered Sensibility of the Pleura—Neuralgia Pectoris—Pleurodynia—Treatment of Nervous Pains of the Chest—Tumors in the Pleural Sac—Signs.

WE shall find no difficulty in comprehending within the present lecture all that needs to be said of other affections of the pleura, not immediately connected with inflammation.

The serous secretion of the pleura may be increased by various circumstances affecting the circulation within the chest, such as organic diseases of the heart and great vessels, tumors at the root of the lungs, especially when these circumstances much interfere with the functions of the organs, as towards their fatal termination. But idiopathic *hydrothorax* is a rare disease; and all that you have heard about water in the chest causing orthopnoea, starting from sleep, lividity of face, &c., belongs rather to the organic disease of the heart than to the liquid effusion, which is often absent with these symptoms, and as we have seen, is present to a great extent in latent pleurisy without producing these symptoms. Hydrothorax is sometimes produced in general dropsical states resulting from febrile action, from a cachectic state of the system, or from diseased kidneys; and when extensive, becomes, together with œdema of the lungs, the cause of oppression and danger in such cases. Slighter degrees of it very commonly take place shortly before the fatal termination of many diseases not essentially connected with the chest, and are probably the consequence of the congestion of blood caused by the gradual failure of the circulation and respiration. In all these cases the effusion may affect both sides; but it is generally more abundant on one side. So also in hydrothorax connected with organic disease in the chest, there is commonly more fluid on one side, and that not always the most diseased. I have frequently remarked that the effusion accompanying organic disease of the heart is most abundant on the right side; this is also often the case when the pericardium is adherent to the heart. The liquid of hydrothorax is simply serum; but in other cases in which great congestion of the thoracic vessels has been its cause, we may often see in it a bloody tinge, or an approach to an inflammatory product, in a few slight loose films of lymph—proofs that the overflow extended also to the nutritive secretion of the membrane, in consequence, probably, of the temporary struggles of reaction which the vascular system frequently displays during the sinking of the vital powers. They resemble the congestive peripneumonies of the dying, which we shall soon have to notice, and should not be confounded with the products of real inflammation.

Now you can readily perceive that the physical signs of hydrothorax are the same as those of the effusion in pleurisy. There

will be the dulness on percussion, and diminution of the respiratory murmur in the dependent parts of the chest, and afterwards you may have ægophony in the middle regions; but as the effusion is seldom so extensive in hydrothorax as in pleurisy, or so much confined to one side, you do not get that abolition of the sound on percussion, and of the respiration and voice, or the displacements of organs, or the peurile respiration on the opposite side, which occur in the latter disease. The general symptoms, exhibiting a dropsical tendency, with effusions in other parts, together with the signs of disease of the heart or other organ affecting the circulation, and an absence of the general, and local signs of inflammation, may generally enable you to distinguish the effusion of hydrothorax from that produced by an inflamed pleura.

The treatment of hydrothorax will vary a good deal with the nature of its cause—from what organic lesion or what constitutional condition it proceeds. Thus, that from disease of the heart will require one set of remedies; that from diseased kidneys another: the former being most frequently relieved by diuretics and hydragogue purgatives, the latter by mercurial aperients and diaphoretics. Again, that connected with a febrile condition of the system, as after scarlatina, may often be removed by an antiphlogistic plan; whilst that arising from a cachectic condition will receive most relief from tonics. To enter into the details of these different lines of treatment would require more time than we can spare, for we have superabundance of matter for the remaining part of the course more immediately connected with diseases of the chest.

Pneumothorax, or air in the pleura, may be produced in three different ways. 1. It may be the consequence of a partial pleurisy. We have before seen that after a pleuritic effusion has long compressed the lung, and the compression has been perpetuated by a rigid false membrane which has been formed over it, the absorption of the liquid leaves a void, which the collapse or contraction of the walls of the chest is in some cases insufficient to obliterate, and this void is sometimes filled with air secreted by the membranes. I have seen two instances of a partial pneumothorax apparently produced in this way. They each occupied about half of the pleural sac—one the upper, the other the lower half; and the lung in both cases was strongly bound down by fibro-cartilaginous membrane, and condensed in the part contiguous to the empty space. There was also some contraction of the chest in both cases. This kind of pneumothorax is very rare; and I do not think that it is described by any author.

2. Another kind of pneumothorax is that which may be called idiopathic, and arises from an effusion or secretion of air into the sac of the pleura without perforation. This is also of very rare occurrence. It is said to be sometimes met with towards the termination of fatal diseases, in the same manner as tympanitis occasionally occupies the peritoneal sac under similar circumstances. I can-

not say that I have ever met with a case in which I discovered any signs of such a form of pneumothorax during life ; although I have several times seen air in the pleural sac on opening bodies after death, where no perforation could be discovered in the pleura. It is possible that the air may have been exhaled from the animal fluids after death, and then increased by exosmosis through the lung. The facility with which air pervades dead membranes countenances such a notion. Pneumothorax is also said by Dr. Graves and others, to have occurred in a few instances at the commencement of pneumonia, and to have soon afterwards disappeared ; but as the chief sign in these cases was a remarkable resonance on percussion, I suspect that these were examples of the production of tracheal or amphoric sound on percussion, from consolidation of the upper lobe of the lungs, and not cases of pneumothorax.

3. By far the most common kind of pneumothorax is that caused by some unnatural communication between the pleural sac and the external air ; and this may be by a perforation either of the external parietes or of the pulmonary pleura. The latter case is the now usually recognised cause of pneumothorax, and constitutes the great bulk of the examples that are met with. The perforation depends on the progress of ulceration, generally of a tuberculous character, rarely of gangrenous abscess, through the pleura. The circumstances of ulceration reaching and perforating the pleura, indicates a low state of the reparative powers, and a want of plasticity in the products of inflammation ; for under ordinary circumstances, ulceration could not approach the pleura without causing it to inflame and throw out coagulable lymph, which, becoming organized, forms either a protecting thickness of membrane, or close adhesions to the costal pleura. We see this in most cases of chronic phthisis, where the upper lobes are generally adherent to the ribs. I have seen an ulceration extend from a tuberculous cavern across the two layers of the pleura, thickened and adherent, and completely through the walls of the chest ; so that when the patient coughed, air bubbled out of two or three fistulous openings in the front of the chest : but here was no pneumothorax. On the other hand, I have met with more than one case in which the adhesive process seemed quite incapable of protecting the pleura, which was consequently perforated in several points ; wherever, in fact, the ulceration of the lung extended to it, and the air freely entered the sac of the pleura by all these holes. More commonly, however, there is only one perforation, and this is generally near the apex of the lung, in connexion with some of the cavities that first form there.

The completion of the perforation is in most instances sudden ; a part thinned by ulceration and imperfectly adherent, giving way during a fit of coughing, or some other unusually forcible act of respiration. The immediate effect is to admit air more or less rapidly into the pleural sac, which by equalizing the atmospheric pressure

outside and inside the lung, permits it to assume that state of collapse to which its natural elastic contractility would reduce it. Hence dyspnœa, sudden and severe, in proportion to the extent to which the air enters and the lung becomes collapsed. But the access of atmospheric air to a serous membrane totally unaccustomed to it, also occasions great irritation and consequent inflammation. Hence a sudden sharp pain and dry cough, with spasms of the intercostal muscles, and a weak, quick, and sometimes irregular pulse. Soon the irritation becomes accompanied with inflammation, and then follow the symptoms of acute pleurisy, with heat of skin and inflammatory pulse; and a liquid effusion is added to the air in the pleural sac.

Now, although a perforation of the pleura will not fail to introduce air into the pleura, the amount and effect of the introduction of this air will vary considerably, according to the size and other circumstances of the ulcerated aperture. If this be very small, or if, as it not unfrequently happens, it be so placed that the walls of the chest close it in expiration, by which it is rendered valvular; or if it be below the level of the liquid, the air introduced by each inspiration will not escape as freely in expiration, and the result will be progressive accumulation of air in the pleura, and a consequent increase of compression of the lung and of the dyspnœa; and in this way perforation of the lung has, in some cases, caused suffocation within a few hours of its occurrence; in others, this catastrophe has been delayed by the vent given to the air by accidental changes of position, or the effect of violent cough, or by puncturing the chest. If the aperture be of larger size, and no impediment occur to the passage of air through it, it will interfere with respiration only so far as it suffers air to pass outside of, instead of into the lung. But when the air passes thus freely, the pleura is more irritated by it, and there is a more copious secretion of liquid, which is generally purulent, and often foetid. In either of these cases, after the subsidence of the spasm first caused by the entry of the atmospheric air, there are no particular symptoms which can serve to distinguish pneumothorax, especially of either of the first kinds. The occurrence of perforation is sometimes denoted by the sudden acute pain of the side, and embarrassment of breathing; which the patient, in some instances, refers to something giving way during a fit of coughing; but such sudden attacks sometimes take place from pleurisy, without perforation; and I have repeatedly known perforation to occur without being followed by any remarkable increase of pain or distress.

The physical signs of pneumothorax are generally very remarkable and distinctive. The presence of air in the pleura will give to the walls of the chest a freedom of vibration, and therefore a degree of resonance on percussion, even greater than that which the air-filled structure of the lung confers on them; so that percussion will give more of the drum-like note or tone which you get by striking over the region of the stomach or of the cæcum. This will be more

marked in proportion as the quantity of air is considerable. The same circumstance will also impair or destroy the sound of respiration ; for the air not only removes to a distance the pulmonary structure where this sound is produced, but also by its pressure diminishes that entry of air in the cells on which the sound depends. There will be, therefore, this remarkable contrast of signs to distinguish pneumothorax—a good or hollow sound on percussion, with little or no sound on respiration, whilst the opposite side gives a worse sound on percussion, but a much more distinct respiratory murmur.

But there is another class of sounds produced in air-filled cavities, which may often give decisive evidence of their existence. Apply the mouth of this India-rubber bottle to your ear, and observe the sound which striking on its outside causes. It is a little tinkling note, like the clink of a piece of metal or glass. This note is an echo produced by the reverberations or repeated reflections of the impulse from the walls of the cavity, and it is shrill and acute, because the reflections are short and quick, in so confined a space. It is the same sort of note that you hear on listening to the mouth of an empty cask, but it is there less shrill, because the space is larger. Any sound proceeding from, or communicated to, the cask, the India-rubber bottle, or any cavity in the body with reflecting walls, will be accompanied or followed by this sort of tinkling or ringing echo, which will be more prolonged and distinct in proportion as the walls are perfectly and uniformly reflecting. You may often hear sounds of this kind, on using the stethoscope over the stomach or large intestines, as their contents move and cause a sound within them. So, too, you may hear the tinkling echo accompanying the sounds proceeding from an air-filled cavity in the chest, and it becomes a distinctive sign of the existence of such a cavity.

In idiopathic pneumothorax, and in that partial kind resulting from the absorption of a partial pleuritic effusion, although the cavity be present, there may be no sound produced in it, or transmitted to it, so as to cause the tinkling echo. Sometimes, percussion on the external walls will do this ; but care must be taken not to strike too near the spot where the stethoscope is applied, or the clink sound produced by abrupt impulses on the tympanum may be mistaken for the tinkling echo. Sometimes the voice or cough may be transmitted to the cavity by the condensed tissue of the lung. I have heard the metallic tinkling accompany both of these in a case of partial pneumothorax, in which there was neither liquid effusion, nor perforation of the pleura. But it is where liquid is present and where the pleura is perforated, that you will most commonly hear the phenomenon of metallic tinkling ; not as Laennec supposed, because these are essential to its production, but because the motions of the liquid, or of the air through the orifice, make sounds within the cavity, which serve to show its echoing properties. So metallic tinkling has often been heard after the operation

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for empyema; showing the presence of air in the pleura, which I have before told you is a very undesirable result of the operation.

Perforation of the pleura, with its consequence, pneumothorax and liquid effusion, is not a very uncommon accident in the course of phthisis, and its physical signs are so remarkable that they can scarcely fail to be recognized even by those who are but moderately versed in auscultation. The tinkling reverberation may present several modifications, which it is useful to notice, as they may serve to give a more accurate knowledge of the condition of the parts, and of their tendencies. When the perforation is small, or obstructed by its position against the walls of the chest, or below the level of the liquid, the tinkling is seldom heard except on coughing or taking a full inspiration, which reaches the cavity, and may throw the liquid into bubbles. The voice may also sometimes find access to the cavity through consolidated portions of lung, and then it will be accompanied by a tinkling. When the orifice is large and free, the air will pass in and out in ordinary breathing, and will produce in its vicinity a sound like that of blowing into the mouth of a glass bottle; and this kind of respiration is therefore called *amphoric*. In such cases there is seldom so much oppression of the breathing as in those where the air passes less freely, and accumulates in the cavity. In listening for the tinkling phenomena, you must recollect that they may be audible only in certain parts of the chest where the lung is not adherent, and where the liquid effusion does not reach. Generally, in the sitting posture, they are heard best about the mamma, and at the lower parts of the scapula; but I have heard them in every part of the affected side, and sometimes only in one spot. In fact, there must be a certain degree of tension in the walls of the cavity to make them good reflecting surfaces; and if this be deficient at the spot opposite to that on which the stethoscope is applied, the sound may be absorbed, and not reflected.

The addition of the liquid to the air in the chest makes the diagnosis still more easy. By percussion you find the exact level which separates the two, and you find that change of position varies the position of the liquid much more distinctly than when there is no air in the pleura. The motions of the liquid also may give very decisive evidence of its presence in the free cavity. On change of posture, or on coughing, the liquid will sometimes drop from the parts which have just been immersed, and the sound of this will exhibit the metallic ringing in so distinct a manner, that it resembles the note which a glass or porcelain vessel gives when struck. If the liquid be agitated more forcibly, as by the patient giving his body an abrupt jerking half turn, it can be heard to splash most distinctly against the walls of the chest; and this is the sign of *succussion* which was described by Hippocrates. This may also be accompanied and followed by the tinkling echo. The splashing is not easily heard unless there be a good deal of air in the pleura, and a certain quantity of liquid. The proportions of these are, however, better to be ascertained by percussion.

I have nothing to say on the treatment of simple pneumothorax. When it is the consequence of the long continuance of a circumscribed pleuritic effusion, no artificial means can remove it ; but if the pleurisy were treated in time it might probably be prevented.

In case of perforation of the pleura, it is generally necessary to adopt measures to relieve the irritation and oppression occasioned by the first access of air into the pleural sac. In the first instance this accident is often accompanied by considerable prostration from the sudden shock to the system, and the pain and cough are rather those of irritation than inflammation. Considerable doses of opium are necessary to allay this pain and the spasms into which the muscles are thrown : it may be advantageously combined with calomel and antimonial medicines ; and sinapisms, or warm fomentations, may be applied to the side ; but more active antiphlogistic measures cannot be used until the reaction takes place, which generally begins in a few hours, bringing with it heat of the skin, strength and hardness of the pulse, and great soreness as well as pain of the whole affected side. Then bloodletting, chiefly local, must be used with aperients and salines, according to the strength of the patient, and the degree of fever present, and followed by blistering, or other means of counter-irritation, in proportion to the continuance of the inflammatory symptoms. But it is not to be forgotten, that perforation of the pleura and its consequences are, in by far the greater number of instances, added to a previously existing disease, tuberculous phthisis ; and the degree of advancement which this may have reached must much limit the propriety and the efficacy of the measures for this accidental inflammation that has been excited. The same considerations are to be held in view when, in consequence of the smallness of the perforation, or its valvular condition, air accumulates in the chest, and becomes a cause of oppressive dyspnoea. The immediate indication in this case is, doubtless, to give vent to the air by puncturing the chest, and this has been done by temporary relief in several examples ; but before this operation is performed, it should be considered whether, as it can give only temporary relief, the condition of the patient be such as to make this likely to outweigh the pain and risks of the operation. These certainly are not great, but when added to the dubious view in which the friends of the patient may regard an operation which proves but imperfectly successful, are enough to deter us from the responsibility of recommending the operation in many cases. The case is different when the accident occurs before the consumptive disease has advanced far, when there is still much flesh and strength, and the physical signs have shown that there is a considerable quantity of sound lung. The operation may be repeated if the air accumulate again. As it is impossible to avoid the continued introduction of air into the chest, the mode of performing the operation is a matter of much less consequence than in empyema. It is more desirable to puncture low down in the chest, to permit the discharge of the liquid as well as the air.

I have only now to say a few words on *altered sensibility* of the pleura and contiguous textures, particularly those nervous pains of the chest which sometimes simulate pleurisy. It not unfrequently happens in sensitive frames, particularly those of females, that an acute pain suddenly seizes some part of the chest, causing shortness of breath, and perhaps cough, very like the stitch of pleurisy. But there is no heat of skin; and the pulse, although often quickened, is not hard. The respiratory motions and sounds may be diminished by the restraining influence of the pain; but the other physical signs of pleurisy are wanting. There is no friction sound or dulness on percussion; but there is sometimes a continued dull rumbling sound, which is produced by the vibrating contraction of the muscles, which is kept up by the sensation of pain. These affections appear to be neuralgic, and are often connected with a condition of the system the very opposite of inflammatory, such as that which comes on after considerable losses of blood, or when the blood is in a poor watery state, as in chlorotic females. I have known them, however, in connexion with irregular menstruation, to occur whilst this discharge was dodging, and to be relieved when it took place. In such cases blood-letting relieves the pain, but at the expense of the natural function. There are other kinds of pain in the chest, which may be called nervous, such as those associated with indigestion or a disordered state of the stomach, which are generally referred to the sternum, and, in the case of gastrodynia, are so severe as to cause great apparent dyspnoea. Pains are also felt in the chest and shoulders from a congested state of the liver. The affection called *pleurodinia* is generally considered to be of a rheumatic character, either in the intercostal muscles, or in the fibrous fasciæ lining the chest. If it occur singly it may be of little consequence; but if connected with constitutional rheumatic disease in other parts, whether accompanied by much fever or not, it is not to be lightly thought of, for it may readily be converted into a pleuritic or pericardial inflammation.

The treatment of the sharp nervous pains in the chest must be directed more to the condition of the system which induces them than to the part thus temporarily affected; for it may be to day the chest, to-morrow the abdomen, another day a limb, that is the seat of these pains. Sinapisms, hot fomentations, and stimulating or anodyne liniments or plasters, will generally relieve the pain. Where the nervous irritation seems to arise from an undue depression or depraved state of the vascular function, as in chlorosis, the careful administration of tonics, especially steel medicines, with due attention to the excretions, will be most beneficial. When the pain seems to be the result of misdirected rather than excessive nervous influence, as in amenorrhœa without chlorosis, those remedies are indicated which tend to draw blood and nervous irritation towards the uterus, such as small doses of aloes, the hip-bath, and for a more continued effect, wearing flannel over the hips and thighs and riding on horseback. If blood is to be drawn at all for tempo-

rary relief, it should be by a few leeches to the inside of the thighs ; and I have known this measure, repeated for several nights in succession, bring on the natural relief when all other means had failed. But these are details which we have not time to pursue further.

I need scarcely occupy your attention with the rare cases of tumors situated in the pleural sac. Enlarged bronchial or thymous glands, and malignant disease originating in them, or in the cervical or axillary lymphatic glands, have been known to form tumors in the chest, especially the upper and anterior parts, pushing aside the lung, and endangering and destroying life by the obstruction which they occasion to the respiration and circulation. Their general symptoms are chiefly referrible to disorder of these functions ; and they are not always accompanied with pain or other irritation, although in some instances their growth is very rapid. They are occasionally accompanied by hydrothorax, or pleuritic effusion. The physical signs which they produce are sometimes puzzling. They are,—dulness on percussion, and abolition of the respiratory murmur, in the parts entirely occupied by the tumor, and sometimes an outward prominence of the walls of the chest at these parts ; bronchial respiration, sonorous rhonchi, and resonance of the voice in other parts, the results of partial pressure on the displaced lung ; or a total abolition of these sounds when the compression is more complete. In such cases we may be aided in our diagnosis by examining the glands in the neck and axilla. If these are enlarged, it is more than probable that the disease of the chest is of the same character. The distinction between these and aneurismal tumors will be considered hereafter.

When of a malignant character, these cases are, of course, beyond the reach of medicine. I have seen two or three cases of tumors in the chest, which, after threatening destruction by their rapid growth, and pressure on the vital organs, subsided under the influence of local bleedings, the internal use of liquor potassæ and hydriodate of potash, and other means calculated to improve the general health.

LECTURE XIX.

Diseases of the Parenchyma of the Lungs:—What is the Parenchyma ?—Pneumonia, its Seat and Causes—General Symptoms—Anatomical History : Engorgement ; Hepatization ; Suppuration ; Gangrene ; State of adjoining Textures—Pathological History ; Functional Symptoms ; Physical Signs ; Cause of crepitant Rhonchus ; Signs of Hepatization, Suppuration and Gangrene—Varieties ; Typhoid Pneumonia with Bronchitis ; Pleuro-pneumonia.

HAVING, as far as our time would permit, considered the diseases of the membranes and tissues investing the lungs, both outside and inside, we have now to think of what lies between these membranes,

the pulmonary parenchyma. But what is this parenchyma, if you take from it the membrane that lines the air-tubes and cells, and the pleura which covers their exterior? See how delicate is the structure of this piece of healthy lung! how thin are the films that separate these minute membranous bubbles—this tissue of froth! How can we distinguish the parenchyma from the investing membranes? We can scarcely do so by anatomy. But physiology and pathology will supply us with a distinction that enables us to make good the division, long acknowledged in practice, of diseases of the lungs into those of the investing membranes and those of parenchyma. Let us recollect the great purpose of the function of the lungs, and we shall bring to mind the air and the blood that exert across the membranes a mutual influence on each other. We have spoken of the vessels which contain the air, but not of those which contain the blood: these it is that lie between the aerial and pleural tissues of the lung, and with the connecting cellular tissue, exceedingly delicate between the cells, but thicker around the tubes and between the lobules, these constitute the proper parenchyma of the lung.

In considering the diseases of the parenchyma, we, as usual, first take up inflammation, that formidable disease *pneumonia*, or *peripneumony*; and there is the greater reason why inflammation, an affection which chiefly concerns the blood vessels, should be a prominent object of study in the parenchyma of the lungs, because this parenchyma is so mainly constituted by blood-vessels. It is well worthy of remark, that the inflammation which we have already considered, bronchitis and pleuritis, affect vessels which freely communicate with those of the parenchyma; but although they do occasionally extend to the latter vessels, and become thus converted into peripneumonic inflammations, yet they are generally quite distinct from this in their present course and ulterior effects, and require a distinction in treatment, which should ever be borne in mind. We find an explanation of this difference in the peculiar character and importance of the pulmonary blood-vessels; in their great number and capacity; in the large proportion which they must bear to the other solids of the lung; and in their great liability to congestive distension. This extensive and important plexus of vessels, through which the whole blood of the body passes, is as we have before seen, peculiarly liable to distension from any cause which may disorder the function of the heart or lungs. Thus all those causes which tend to induce asphyxia, produce also that congestion of the pulmonary vessels which, added to irritative reaction, may constitute inflammation; and thus we see that various causes which affect the balance of the circulation, particularly by deranging the passage of the blood through the lungs, such as asphyxiating poisons, congestive fevers, violent exertion, diseases of the heart, bronchitis, asthma, tubercles in the lungs, long-continued exposure to cold, and the moribund state, may become causes of pneumonic inflammation.

It is scarcely necessary to detain you with a notice of the general symptoms of pneumonia. You will find them amply described by authors ; and I shall be the less particular in stating my own views on the whole subject of this disease, because what I have to say more than is to be read in books, may, for the most part, be found in the article "Pneumonia," in the *Cyclopædia of Practical Medicine*. With respect to the general symptoms, I dare say that your own experience has suggested to you how insufficient they sometimes are to characterise the disease. The pain in the chest generally more dull and deep-seated than in pleurisy, the cough with more expectoration, the pulse quick, but often soft ; the quickened, and sometimes very oppressed breathing, made worse by lying on the affected side, often truly indicate the complaint ; but the pathologist knows that these symptoms are more accidental than essential, and that they may all exist without pneumonia. Examinations after death have often startled medical men by discovering proofs of a pneumonia, the existence of which had scarcely been suspected during life ; and did time permit, I could cite many instructive anecdotes to this effect. But now let us resort to the anatomy of the disease, to instruct us as to the physical changes which pneumonia produces ; and we shall then be enabled to understand the physical signs that they may produce during life.

The first condition produced in the lungs by inflammation is that of *sanguineous congestion* or *engorgement*. The vessels are then so much distended that the whole tissue appears red, of different shades. In some cases a frothy serum exudes when the lung is cut: this is probably the effect of the coagulation of the blood after death, for it is not observed in those cases in which the blood remains in a fluid state. It is a common notion that the blood of an inflamed lung is effused into the air cells ; but several reasons incline me to believe it to be contained still in distended vessels, and in the tissue, although it may afterwards be extravasated. Andral examined an inflamed lung after drying and slicing it ; and the only difference which he could perceive in the structure, was, that the coats of the cells were somewhat thicker and redder than natural ; but there was not that obliteration of the cells that might have been anticipated if they had been filled with blood. In typhoid pneumonia, and that from asphyxiating gases of the sedative kind (such as those of sewers), the inflammation does not seem to go beyond this stage ; or if it do, it passes at once into a half gangrenous, half purulent destruction of the tissue, there being, apparently, under these circumstances, some change in the vital properties of the tissues, or in the condition of the blood, which incapacitates it from supplying lymph, the deposition of which constitutes the next stage, that of hepatization.

The second stage of inflammation, *hepatization*, when complete, brings the lung into a state of solidity like that of liver. But you are not to suppose that the transition from the first to this stage is sudden ; it is gradual, and is the result of that same overflow of the nutritive function which we found to cause the effusion of lymph

on the inflamed pleura. The tissue of the lung, besides being rendered more solid, is also generally more fragile than usual, so that on being pressed it breaks down under the finger. This softening is, I think, chiefly the consequence of the interstitial deposit of soft fresh lymph, which diminishes the molecular cohesion of the tissues; and the more acute and recent the inflammation is, the greater in general will be the softening. The colour of a hepatized lung will vary much, according to the quantity of the blood left in it; if this be much, it will be red; if little, pinkish brown or reddish grey, if mixed with the black pulmonary matter. The deposition of lymph seems to supersede the red particles, or it may possibly be formed at their expense. A hepatized lung, on being cut or torn, often presents a granulated aspect; numerous little points, of the size of pin-heads, being of a lighter colour than the rest. These granules Andral first represented to be the single air-vesicles, or terminations of the bronchi; and he conceived that they were distended with the same viscid mucus, secreted by their mucous linings, which is seen in the sputa. In this opinion he was joined by Laennec. Soon after this view was published, I made minute examinations of the granulations of hepatized lungs, and was soon convinced that they contained no appreciable quantity of the viscid mucus of the expectoration; and I was led to regard them as vesicles with their coats distended by an interstitial deposit of lymph, and perhaps containing the same matter in their interior. In his more recent work on Pathological Anatomy, Andral has expressed a similar opinion. But hepatized lungs do not always present this granulated appearance; sometimes there is a uniform condensation of a deeper red than usual; and this condition Andral refers to a more complete obliteration of the cells, a further degree of solid effusion. But this would not account for the redder colour; and I would rather view this kind of non-granular hepatization as the result of inflammation confined more to the plexus of vessels and interstitial tissue, and affecting less the membranes forming the cells; hence the consolidation partakes more of the character of the vessels and of the blood which they convey, and less of the lighter coloured deposit which the membranes of the cells secrete.

The third stage to which inflammation brings the lung is that of *suppuration*, or *yellow hepatization*. This consists in the conversion of the semi-solid particles of lymph or blood, which constitute the solid of red hepatization into an opaque, light yellowish, friable matter, and finally into a fluid pus. This suppuration is generally diffused in the form of purulent infiltration; and it is very rare to find it assume the character of a distinct abscess. You see a sufficient reason for this in the very porous structure of the lung, which renders the circumscription of the matter, by the effusion of lymph, such as takes place in abscesses in general, an unlikely result; and the life of the patient, as well as the vitality of such a delicate and porous structure as that of the lung, is generally destroyed before the process of suppuration can be completed. Hence, even

where the suppuration has advanced most, there is generally much of the tissue of the lung remaining; and a gangrenous condition is often added to the suppuration, giving the matter a very offensive odour. Nevertheless, circumscribed abscesses are now and then met with in the lung; and this is generally when the inflammation is limited, or more intense at one part, so as to lead to the early formation of pus, whilst the adjoining parts are still capable of throwing out a circumscribing lymph. In this way I have met with abscesses terminating pneumonia affecting separate lobules; thus, too, abscesses are formed around foreign bodies within the lung, such as a musket-ball; and around calcareous and scrofulous tubercles, which may act as foreign bodies. In all these cases the inflammation of the most irritated parts reaches the stage of suppuration long before that of those around them, and the latter thus forms a separating wall of effused lymph, which may afterwards constitute a kind of cyst. The purulent deposits which are sometimes met with in the lungs of patients who have had extensive suppurations in distant parts, generally present this circumscribed character.

Gangrene, unconnected with suppurations, is a very rare sequel of inflammation. It seems, however, to arise pretty generally from the influence of those noxious gases which directly destroy the vitality of the tissue of the lung. The lungs of persons who have died some days after being nearly asphyxiated in sewers, have been found reduced in parts to a dark brown, greenish, or livid softening; having a very fœtid odour, and being probably the result of the poisonous influence of the gas on a congested lung.

The state of the tissues adjoining the vascular plexus, the proper seat of pneumonia, is worthy of notice. The interlobular cellular texture sometimes partakes of the general redness, and sometimes it is singularly free from it, or has it so much less, that a section of the lung is quite marbled by its lines: so also in the hepatized stage, the interlobular septa retain their cohesion, and, in more chronic cases, sometimes become thicker and harder than usual. The mucous membrane of the large and middle-sized bronchi is almost always more or less inflamed, and presents the red striated aspect that is also seen in the more intense forms of bronchitis. That of the smaller bronchi is often of a deeper red than in bronchitis; but, from its bluish tint, this would appear to arise rather from the blood under it than in it. The bronchi in the inflamed part generally, but not always, partake of the softening in the parenchyma; and, in the stage of hepatization, I have sometimes seen the finer ones plugged with lymph—conditions which I have before noticed to you as likely to lead to the permanent obliteration of some tubes, and the dilatation of others. More commonly, however, the tubes contain more or less of the slimy rusty mucous which is seen in the expectoration. The pleura is generally, but by no means constantly, inflamed; at least I have seen it free from redness, lymph, and liquid effusion, even when covering a hepatized lung. The cases of pleuro-pneumonia in which the inflammation of the pleura is so

considerable as to modify the course of the pulmonary inflammation, will require our attention afterwards.

Now let us see what effects these various lesions will have on the vital and physical properties of the lungs, and in developing symptoms and signs of their presence. The disorder of the vital functions need scarcely occupy our attention, for they are amply described in works; and although often most important in directing our treatment, it frequently fails in distinguishing pneumonia from other affections of the organs of respiration. The febrile disturbance is commonly of a very pronounced kind, as it might be expected to be from the extent of the vascular plexus involved in diseased action. But it is very uncertain whether this fever shall be of a sthenic or an adynamic type, for we meet with pneumonia of both characters; and we find it also variously complicated with febrile irritation, particularly involving the hepatic and gastric functions. We meet with it also associated with the various eruptive fevers, and arising out of the congestions left by remittent and continued fevers of the typhoid type. You must see that our time will not permit us to enter into the details of these varieties, which are more proper subjects for a course of clinical medicine; and I shall only say a few words on the general symptoms most common to pneumonia in general. The pulse is generally much quickened, and in the early stage it presents often the strength and hardness common in severe inflammation. But the disease in its severe form seldom extends far, or continues long, without a change in the pulse, which, retaining and increasing its quickness, becomes small and thready, or liquid yet jarring, as if a little blood were thrown with much force into a large and loose vessel. Such, in fact, is the case, for the obstructed state of the lungs prevents the left side of the heart from receiving its due supply of blood, whilst the right side is over distended, and irritated into increased frequency and abruptness of contraction, which are communicated to the left. Whether these pulses shall reach the radial artery as small thready strokes, or as looser liquid ones, will depend on the condition of the surface and the tonic contractility of the artery; circumstances that I formerly adverted to in speaking of the analysis of the general symptoms of diseases of the chest. Hence you can understand that the pulse may be very deceptive with regard to the amount of the inflammatory disease; hence, too, it is not at all unusual to find it increase in fulness and force after blood-letting. The difficulty of breathing is frequently by no means proportioned to the extent of the disease; although, when present, it is less equivocal than the pulse, and may be a better guide to distinguish the variations of the complaint. Cough is a very uncertain symptom, being often very slight in the worst cases, and, as may be supposed, depends rather on the attendant bronchitis than on the parenchymatous inflammation itself. But the cough, however slight, is generally accompanied by the expectoration of a matter, which is in great degree characteristic of pneumonia, and this circumstance makes it a symptom of much

importance. The sputum of pneumonia is that of the most intense form of bronchitis, viscid, glutinous, capable of being drawn into threads, and having at first a certain degree of transparency, but afterwards often becoming opaque; but what distinguishes it from that of bronchitis is its reddish or rusty colour, which sometimes passes into an orange, a yellow, and even a greenish tint. These colours depend on the intimate admixture of different small proportions of the colouring matter of the blood, and we cannot doubt that it proceeds from the mass of blood accumulated in the pulmonary plexus, and tinging the bronchial mucus. This rusty expectoration was first described by Andral, and it is considered by him and others to be quite pathognomic of peripneumony. To this, however, I cannot assent; the rusty tinge may be communicated to the bronchial mucus by congestions that are not inflammatory, and I have seen an expectoration of the same character in cases of bronchitis supervening on organic diseases of the heart causing great pulmonary congestion, and also on pulmonary apoplexy: either of these complications may be attended by the expectoration of viscid semi-transparent mucus, variously tinged with the colouring matter of the blood. Still, when occurring with other symptoms of inflammation of the lung, these sputa may be considered as pretty surely conclusive of its existence. The intensity of the inflammation is sometimes represented by the degree of viscosity of these sputa; but this is not always the case; nor can you expect it to be so, when you reflect that the sputa are the product, not of the pneumonia, but of the bronchitis attending on the pneumonia, which are by no means in constant proportion to each other. So also it sometimes happens that the expectoration is altogether wanting; and in the case of young children it is seldom brought within our view.

If we study the physical signs of pneumonia, we shall find in them much surer means of distinguishing the disease, and they will furnish us with a good pathological history of the conditions of the affected organ. On listening to the chest of a person with incipient inflammation of the lungs, you will hear, generally in the inferior and posterior region of one side of the chest, a fine crackling sound accompanying the respiratory murmur. In its slightest degrees it is scarcely more than an unusual loudness and roughness in the vesicular murmur, as if the air met with slight short resistances in its passage, which destroy the smoothness of the sound; but in its more pronounced degree there is a distinct crepitation, like that heard when kitchen salt is thrown on a hot iron, or like that caused by rubbing between the finger and thumb a lock of hair near one's ear. It is first heard at the commencement of inspiration and the end of expiration; but it soon accompanies the whole respiratory act, and in advanced degrees of the first stage it is heard only at the end of inspiration and the beginning of expiration.

Now what is the cause of this fine crepitation, or *crepitant rhonchus*, as it is also termed? M. Andral considered it to be produced by the passage of air in minute bubbles through serum effused in the

minute air-tubes and vesicles, and that the fine and even character of the crepitation depended on the smallness of the bubbles in the extreme tubes. Thus he supposed that this crepitation differed from the mucous rhonchus only in the size of the tubes in which it is produced, and the consequent size of the bubbles in them. This opinion has been adopted by most writers; and you will find the various liquid rhonchi of catarrh, and the crepitation of pneumonia, all reduced to the two heads—large and small crepitation. From a consideration of the pathology of pneumonia, and the course followed by its physical signs, I was long ago led to consider the crepitation which attends its early stage distinct in nature from the other rhonchi. We have before had occasion to notice that the structure and motion of the lungs tend to bring all liquids secreted in the minute tubes into those of larger size, whence they are ultimately collected in the trachea, and expelled by expectoration. Now were the crepitation of pneumonia dependent on serum in the smallest tubes and cells, we ought to have proofs of the presence of this serum in the other tubes by a bubbling rhonchus, if not in the expectoration as well. Now I have had frequent occasion to observe that the first stage of pneumonia is in many cases remarkably free from bubbling sounds in the large tubes; and you know that the expectoration is of a viscid mucous, instead of a serous character. In fact, there is good reason to believe that the serum which exudes from an engorged lung after death, and which Andral assumed to be the cause of the crepitation, is chiefly the result of a cadaveric change, the coagulation of the blood in the distended vessels, which does not take place during life. There are other reasons, which we have not time to state, which induce me to take another view of the cause of the crepitant rhonchus.

What is the condition of the extreme air-tubes and cells in the first stage of peripneumony? Are they not narrowed and partially obstructed by the enlarged vessels which are distributed between and around them? And as the smallest tubes are narrower than the cells in which they terminate, may not the obstruction become such in them, that the air can force itself through the viscid mucus which lines them only in successive minute bubbles, the crackling of which constitutes the crepitation in question? This appears to me to be the true mechanism of the crepitant rhonchus; and in this view you can understand how, at first, the crepitation must be but slight, and confined to the most collapsed state of the tissue; that as the narrowing increases, it extends to the whole respiratory movements; that subsequently the obstruction is such that it permits the crepitating passage of the air only when the lungs are most expanded, as at the end of inspiration and the beginning of expiration; and finally, that the obstruction becomes complete, and the crepitation ceases, except perhaps still, on a forced inspiration.

You can see, too, that the natural respiratory murmur will be rendered rough, and perhaps sharper, before the crepitation be-

gins* ; that it will be diminished as the crepitation comes on and extends to more of the minute tubes ; and that it will cease when the abnormal sound occupies them all. If the disease be extensive, and the function of the lung much infringed on, the energy and frequency of the respiratory movements will be increased, and consequently the respiratory murmur on the sound side will be louder than usual, having the character of *puerile* respiration.

The increased matter of the congested lung will have further effects on its properties with regard to sound. It will deaden the sound on percussion, so that the affected side will give a sound rather duller than the opposite side ; and different degrees of force in percussion will not materially affect this variation. But the first stage of inflammation, without liquid effusion, is insufficient to make the sound on percussion quite dull ; for even in its most advanced degree there is still enough of air in the lung to give an elastic resistance to the walls of the chest, and to leave their vibrations pretty free. The motion of the affected side will be diminished in proportion as the air fails to get admittance into the inflamed lung ; and instead of being fixed in a state of distention as in pleurisy, or in a state of contraction as in spasmodic asthma, the side holds an intermediate size, measuring less than the sound side on full inspiration, and more than it on the completion of expiration. The increased density of the congested lung also makes it conduct sound better than in the light spongy condition of health ; so that in extensive inflammation, even during the first stage, and whilst the crepitation yet continues, you may hear in some measure the bronchial respiration and vocal resonance, that are fully developed only in the stage of hepatization. Let us pass on to the signs of this stage.

The deposition of lymph which constitutes hepatization of the lung completes the obstruction of the minute tubes and cells : hence all crepitation ceases, and the only sounds that reach the ear are those of the air and voice in the larger tubes. The respiration is no longer heard with its prolonged murmur ; but in the neighbourhood of the bronchial tubes only there is a *short whiffing*, confined to parts only of the respiratory act, and often ending abruptly with a *click*. This bronchial whiffing is not to be heard in every case, but only when the hepatization involves considerable bronchial tubes, and is most commonly found in the middle portions of the chest. Here, too, may be both heard and felt the various degrees of morbid bronchophony, or vocal resonance. When the consolidation of the lung is very complete, and involves especially the central parts of the lung, the voice may be heard to resound over a

* Dr. Stokes considers that there is a stage of pneumonia before the crepitation begins, and that it is denoted by a puerile, or unusually loud respiratory murmur in the part. This is an important observation ; but I doubt the propriety of calling that another *stage*, which probably is only a smaller degree of the first stage, and which afterwards causes crepitation—the partial narrowing of the tubes.

space of considerable extent in the mammary, scapular, or axillary region, and so loudly, that it appears to issue from the part like the voice in pectoriloquy, with which it is sometimes confounded. You may generally distinguish the bronchophony of a consolidated lung, by observing that it extends over a considerable space, and that it is greatly diminished by using the stethoscope with the stopper in, which is not the case with the pectoriloquy of a cavity. The vocal resonance of the tubes is also transmitted by the condensed lung to the parietes as a vibration, or *fremitus*, which may be distinctly felt by the hand placed on the affected side, and which is much stronger than that on the healthy side. This affords an easy mode of distinguishing between a hepatized lung and a pleuritic effusion; for the latter generally abolishes completely the vocal vibration. The dullness on percussion is now pretty complete, but it is seldom so uniform and general in the lower and middle portions of the chest as the dullness from liquid effusion. You will see as a reason for this, on examining a hepatized lung, that there are generally some lobules or parts retaining enough air to prevent them from sinking in water; and this is also sufficient to prevent the sound on percussion from being perfectly dull. And when the consolidation is perfect, it will transmit the stroke of percussion to deeper seated parts, which will yield their resonance. Thus on the left side a hepatized lung will sometimes give you the tympanitic resonance of the stomach; and near the sternum, or in the mammary regions, you may occasionally have the amphoric resonance, or *bottle-note* of the large air-tubes. It is not difficult to distinguish these sounds from that of healthy percussion, and when once you understand their cause, their presence and properties will serve rather to instruct than to confuse you. In the hepatized stage, the lung being inextensible, you have the corresponding walls of the chest nearly motionless; and they are so in a state neither of distension nor of contraction, without fulness of the intercostal spaces, or displacement of the viscera, and thus you have further distinctions between this case and that of pleuritic effusion. When the left lung is solidified, it transmits the sounds and impulse of the heart to an unusually wide extent of surface, instead of diminishing and displacing them, as pleuritic effusion does. When the hepatization is complete, and involves the root of the lungs, I have known it to cause a bellows murmur at each pulse, over a considerable extent of the affected side; and I think that a similar phenomenon has been described by Dr. Graves, of Dublin. This is probably owing to a partial pressure produced by the solid deposition, on the pulmonary artery, or some of its chief branches, and then transmitted with the current throughout the consolidated lung.

In the third or suppurative stage there is no change in the condition of the lung which can modify the physical signs until the effused matter begins to liquefy; and then there is a mucous or bubbling *rhonchus*, from the secretion into the air-tubes. There may be a simultaneous change in the nature of the expectorated matter,

if there be strength enough left in the body to keep up this excretion. Andral describes it to be sometimes a thin slightly glutinous liquid like treacle and water, or the juice of stewed prunes: this is a sero-mucous fluid, coloured with the hematin in an altered state. I have seen it more after pulmonary hemorrhage than in pneumonia. I have seen pus expectorated in the third stage of pneumonia without abscess; but I believe more commonly there is no expectoration at all, or such only as comes from the upper tubes, and therefore gives no evidence of the state of the lung. In fact, we are rather to infer the supervention of the third stage from the duration of the disease, and the general symptoms, than from any physical signs. The inflammatory symptoms and fever give way to great prostration, rigors, cold sweats, a weak thready pulse, whilst the breathing is as short as ever, and the countenance exhibits the pallid, waxy, anxious, drawn, tremulous features of ebbing vitality.

The formation of abscess is less unfavourable, because it implies less extent of suppuration, and a power in the structure to circumscribe it. There are several cases of recorded recovery from pneumonia after the signs of abscess had manifested themselves. These signs are, those of a cavity, first containing liquid and air, and the seat of a coarse bubbling or gurgling sound on coughing; and after the expectoration of pus, cavernous or hollow respiration, with pectoriloquy, or loud resonance of the voice, in some part of the chest corresponding with the affected spot. These abscesses are not uncommonly of a gangrenous character; and then there is added to the signs a putrid factor in the matter expectorated, as well as in the breath of the patient.

Now without entering into further details (for which I must refer you to my treatise in the *Cyclopædia*), you may perceive that the physical signs of pneumonia will indicate the situation and extent of the inflammation; and, as far as relates to the diseased organ, they may guide us in the prognosis and treatment. Thus a crepitant rhonchus heard in a whole lung, or in a considerable part of both lungs, implies extensive disease. If heard at the root at the lung, or at its apex, that is, at the scapulæ, or under the clavicles, it indicates a severer form of disease than if heard only at the lower parts of the lung in the back. The extension of this crepitation, or its presence, in new parts, is a proof of the increase of the inflammation. Its cessation, and the substitution of bronchial respiration, are proofs of its advancement to the second stage. On the other hand, the return of the crepitation where it had been replaced by bronchial respiration, and perfect dulness on percussion, announces a progress towards cure, by the absorption of the obstructing lymph, and by the air again gaining a straitened admission into the air-cells. As this process proceeds, the act of respiration, accompanied by crepitation, becomes longer in duration, until it equals the duration of the respiratory movement on the healthy side; but the sound is still for a time somewhat whiffing where it has been bronchial; and a crepitation of a looser, less even character, also remains after ap-

parent cure, this is the *sub-crepitant* rhonchus, and probably depends on the presence, in the smallest bronchi, of a little thin serous mucus, such as that which is seen in the expectoration, and the secretion of which seems, as in bronchitis, to assist in removing the depositions left by the inflammation. If the inflammation have proceeded to the stage of hepatization, and particularly if it have verged on that of suppuration, in which the albuminous deposit, becoming opaque and lower in vitality, is less susceptible of absorption, the restoration of the texture of the lung to its natural light condition requires a considerable period of time, even after the apparent cure of the disease ; and during this period there will remain more or less of the physical signs just mentioned, as well as some dulness on percussion, and perhaps, also, not a full power to expand that portion of the chest. We suppose here the cure to become perfect in time, but there are other cases, in which inflammation of long duration produces permanent changes in the lung, by the entire obliteration of some portions of the tissue, and the dilatation of others : these changes happen most frequently when the inflammation is modified by a contemporaneous effusion in the pleura. This we shall notice hereafter.

We have not time to examine all the varieties and complications which inflammation of the lungs presents in its signs and course ; but there are two or three which I cannot pass over.

The form of pneumonia that is called typhoid, whether it be secondary to continued fever, or whether it be primary and assume the typhoid type, from a constitution lowered by excesses, or by the depressing influence of foul air or of an unhealthy season, differs remarkably in many of its phenomena from the common pneumonia. The local symptoms are by no means prominent, and although there may be pain, cough, and very imperfect breathing, the obtuse state of the mental faculties prevents attention from being drawn to them. But the general functions are greatly disordered ; the pulse is quick, small, and weak ; the skin is harsh, dry, and partially hot, or covered with a clammy sweat, and sometimes spotted with petechiæ ; the tongue is furred, brown, and dry ; the alvine excretion dark coloured and otherwise disordered ; and the urine is scanty, turbid, and ammoniacal. The lungs in such cases are sometimes found after death so engorged, particularly their posterior portions, that they sink in water ; the texture is very soft and fragile, and when broken exudes a dark grumous blood : there is only an imperfect approach to hepatization, but the texture in some parts occasionally shows a softening of a lighter colour, which seems to be an imperfect suppuration. In these cases we find dulness on percussion, and absence of the vesicular respiration in the posterior parts of the chest : but no crepitation ; or if it be present, it is of very short duration. In the anterior parts of the chest the breathing may be distinct enough, and accompanied by sibilant and sonorous rhonchi. Now I cannot help viewing these cases as of a congestive rather than of an inflammatory character. By some unknown cause, whether in the condition of

the blood, or in the affected capillaries, or in both, the blood stagnates and accumulates in particular viscera ; generally to some degree under the influence of gravitation ; and the functions of the organs are proportionately impeded or disturbed. There is at the same time more or less irritation, which may give this congestion somewhat of an inflammatory character ; but its products are imperfect and irregular ; and neither in the effusion of lymph, or in the formation of pus, is there manifest a true inflammatory orgasm. We can see why, in the lung, this condition should be unaccompanied by the usual signs of the gradual formation of a crepitating obstruction, because the engorgement is at once produced, and renders a great part of the tissue impervious to air. But this degree of congestion, if it occupy the middle parts of the lung, may give bronchophony and bronchial respiration.

The complication of pneumonia with bronchitis is very common ; in fact, as I have before said, there is bronchial inflammation in nearly every case of pneumonia. But it sometimes happens that pneumonia becomes added to extensive bronchitis ; and this, especially when attended with copious secretion, obscures the physical signs of the pulmonary inflammation. On applying your ear to the chest, you hear sonorous, sibilant, and mucous rhonchi, so loud and general, that you can scarcely distinguish whether crepitation be present or not. In such a case listen particularly to the end of each inspiration, at the inferior and posterior regions of the chest, about the margins of the lobes, and if crepitation is to be heard at all, it will generally be then and there. Look out also for the rusty tinge in the sputa, and as the disease proceeds, for greater dulness on percussion ; and you will generally succeed in discovering by these means when the inflammation has extended to the pulmonary plexus of vessels.

The effects of a concomitant pleurisy on the pathology and signs of pneumonia, are more remarkable. Whenever the inflammation extends from the lung to the pleura, it may be supposed to increase the serous secretion, and perhaps lead to the effusion of lymph ; but when the pulmonary inflammation has existed first, and become extensive, these pleuritic products are commonly to small amount. When the inflammation has simultaneously attacked both the parenchyma and the investing membrane, and nearly to an equal degree, constituting the disease called pleuro-pneumonia, the effusion of the pleura by its pressure modifies the effect of the inflammation in the lung. The lung is found after death consolidated, but tougher and redder than in the state of ordinary hepatization, and totally destitute of the granular aspect. It very much resembles the substance of muscle ; hence Laennec termed this condition *carnification*. It seems to exhibit the more essential part of inflammation of the lung ; the sequel or effect, effusion into the coats of the air-cells, which constitutes granulation, having been prevented by the pressure of the external liquid effusion. This combination is therefore, too, slower in progress than simple pneumonia ; the degree of

the inflammation, as well as the quantity of its product, is restrained by the external pressure, and it scarcely, if ever, proceeds beyond the second stage. But this slower rate of process tends to make its results more permanent. If false membranes are thrown out on the pleura, they become more firmly organized, and bind down the lung in its more compressed state; and the lymph effused in the tissue itself, scanty though it be in comparison with that of a hepatized lung, may become the means of adhesion of the sides of the compressed cells and finer tubes, and of consequent obliteration of more or less of the proper tissue of the lung. When the liquid effusion is removed by absorption, and when the chest becomes again capable of expansion, what will supply the place of the obliterated cells? There will be the atmospheric pressure at each inspiration brought to act on the obstructed tubes; the air can no longer penetrate to the cells; so it must dilate the tubes, and make their increased size compensate for their defective terminations. Pleuro-pneumonia is, then, as I have before had occasion to remark, a cause of dilatation of the bronchi; and, in stating this, I have better ground than theory, from having watched two remarkable cases soon after their inflammatory origin in acute pleuro-pneumonia, through a chronic course, in which signs of permanent consolidation of the lung, dilatation of the bronchi, and contraction of the chest, were the physical conditions which accompanied dyspnoea, weakness, and a dropsical and cachectic state, which ultimately terminated in death, when I found on dissection the conditions which I have described.

On referring to the records of other cases of dilatation of the bronchi, I have met with several in which the symptoms are described to have originated in an inflammatory attack, like pleuro-pneumonia; and I incline much to the opinion that all the cases in which the dilatation affects the bronchi of one side only, and in which there is also general consolidation of the lung, with some contraction of the chest, owe their existence to this cause.

The signs of pleuro-pneumonia are a combination of those of pneumonia and of liquid effusion in the pleura. At first there is crepitation; but this, as the lung is pushed aside by the liquid, becomes indistinct, whilst the dulness on percussion is much more marked than in pneumonia, at least in the lower parts of the affected side. In the central region of the chest, bronchial respiration and bronchophony are soon produced by the condensed lung being pushed against the walls; and if a thin layer of liquid intervene, the bronchophony acquires a loud buzzing accompaniment, like the voice of *Punch*; in fact, the voice seems double, which probably depends on a part of its vibrations being modified into a buzzing or bleating, by passing through the thin layer of liquid, whilst other vibrations pass unchanged. The vocal resonance is generally louder in pleuro-pneumonia than in either pleurisy or simple pneumonia; and I suppose this arises from the chief tubes being pressed so closely against the walls of the chest, with complete condensation of the vesicular structure. The same circumstance will sometimes give

the amphoric or tracheal sound on percussion in the mammary region, which forms a singular contrast to the dulness of other parts : so also I have found the respiration quite tracheal in this spot, and the resonance of the voice quite as loud as that of caverns.

LECTURE XX.

Diseases of the Parenchyma of the Lungs (continued)—Treatment of acute Pneumonia—Treatment of Typhoid Pneumonia, &c.—Chronic Pneumonia—Anatomical History—Pathological History, Signs and Treatment—Œdema of the Lung—Pulmonary Hemorrhage, or Apoplexy ; Causes—Anatomical History—Signs and Treatment—Pulmonary Emphysema, or Dilatation of the Air-cells.—Anatomical History ; Tense and Flaccid Emphysema—Causes, and Pathological History—Signs of Tense Emphysema—Signs of Flaccid Emphysema—Interlobular Emphysema—Treatment of Emphysema.

I SHALL say but little on the treatment of pneumonia, for you will find this subject pretty fully given in the article in the Cyclopædia. It will be enough to give you a word or two on the principles which may guide us in the application of remedies, as far as I have found by experience that those principles hold good.

Seeing that distension of the great pulmonary plexus of blood-vessels is the first condition of pneumonia, from whatever cause it may proceed, we may hope, in the early stages of the disease, to relieve this distension, as well as to prevent the process of reaction which renders it inflammatory, by free blood-letting ; and in some instances, where the local signs announce the presence of the disease in its first stage, and the general symptoms prove it to be of a distinctly sthenic character, the free loss of blood by one venesection will cut short and completely cure it. But when the vessels have been so long distended, and become so much the seat of irritation that the mere removal of pressure from the sanguiferous system at large will not enable them to recover their usual size, a single blood-letting will not be sufficient ; it must be repeated as often as the strength will bear it, and other remedies must be used, which in another way countervail the inflammatory irritation. Of these the most important are tartarized antimony and mercury.

The tartarized antimony is, according to my experience, the most powerful remedy in the sthenic forms of inflammation, especially in their earliest stage. Its mode of action is not well understood. It certainly does not subdue inflammation merely by its nauseating, emetic, purgative, or diaphoretic effects ; for although these modes of operation are occasionally induced by it, yet it is often quite as successful, without being attended by any of them. It appears to be directly antiphlogistic, and we can only conjecture that it is so by a specific action on the inflamed vessels ; which, after all, is just as intelligible as a specific action on the vessels of the intestinal

canal, the liver, or the kidneys, which we ascribe to purgatives, mercurials, and diuretics. I cannot suppose that its operation is merely that of a strong counter-irritant, inflaming and causing pustulation of the gastric mucous membrane, as the Broussaïans maintain ; for although there have been two or three examples in which such effects have resulted from its use, these are extraordinary cases; and among very many instances in which I have seen it used to a great extent, I have never met with any in which, after due discretion in its administration, any symptoms of permanent gastric irritation have continued during its use. In the wards of Laennec, I have seen patients taking as much as thirty or forty grains of tartar emetic daily ; not only without causing sickness, redness of the tongue, pain, or diarrhœa, but sometimes even without destroying the appetite for food, or the power to digest it. Such a quantity as this however, is unnecessary ; and I believe that all the good effects of this remedy may be obtained from twelve grains, or less, in the twenty-four hours. I commonly give from one to two grains in an ounce and a half of some nicely-flavoured liquid, every second, third, or fourth hour, according to the severity of the case. The first dose generally causes vomiting, but this generally ceases with the second or third ; or if it should not, a drop of hydrocyanic acid, given after or with the draught, will often stop it. So also purging may be restrained by the addition of an opiate. But for these details I must refer you to the works of Laennec and Stokes, and to the article in the *Cyclopædia* before named.

Mercury is better adapted to the less sthenic forms of pneumonia, and to its second stage, after blood-letting has been used as far as it can be with advantage. The treatment of the second stage, hepatization, which is sufficiently announced by its physical signs, must be conducted on a different principle from that of the first. There is then solid matter already effused, and no blood-letting, or other means of depressing the circulation, can remove this. Their utility is therefore much more limited, and is chiefly confined to the removal of any increase of irritation or congestion, or to reduce the bulk of the blood to the capacity of the abridged state of the respiratory organs. The chief remedies are those which modify the action of the extreme vessels, and promote the absorption and elimination of the effused matter. Mercury is the most powerful of these, and calomel is the most convenient form ; and its operation may be materially aided, and troublesome symptoms allayed, by combining it with opium and using an alkaline expectorant mixture, varied in composition according to circumstances. It is at this period, too, that blisters and other counter-irritants become useful ; and if the disease has not reached the stage of hepatization, they seem to assist in restoring the healthy condition of the pulmonary vessels.

There is only one recommendation more, which I think it necessary to make, beyond the usual rule of spare diet in pneumonia ; and that is, of as much abstinence from liquids as is consistent with the comfort of the patient. I am convinced that no temporary

cause tends more to increase the dyspœna than distending the stomach, and consequently the blood-vessels, by large draughts of liquid, whether of tea, barley-water, or any other neutral beverage; and the relief given to the congested vessels by blood-letting is often for a time frustrated by the copious libations in which patients indulge soon after it. A total abstinence from liquids is scarcely practicable or desirable; but great moderation in their use, by sipping now and then, rather than drinking, is both practicable and beneficial. You may suppose that this advice is of a piece with that which I gave for the treatment of a catarrhal cold; but it is not. I cannot claim to myself originality in this;* for I heard Professor Hamilton, of Edinburgh, sixteen or seventeen years ago, strongly recommend abstinence from liquids in pneumonia; and so much was he convinced of the propriety of this plan, that he even fancied that pediluvia sometimes do harm, by supplying liquid through the means of cutaneous absorption.

The typhoid form of pneumonia requires a considerably modified treatment. Blood-letting not only is very ill borne, but it appears to have little influence on the disease. There is, in fact, no vascular tension or tonicity; and you would empty the great blood-vessels and stop the heart's action before you could relieve the congestion of the lungs, or enable their vessels to contract. The depressing influence of typhoid or adynamic diseases renders any loss of blood hazardous; and local depletion is the utmost that can be attempted. Considerable advantage may, under these circumstances, be sometimes obtained from dry cupping on the chest, which, for the time, tends more effectually than even blood-letting, to draw the fluids from the congested organs, whilst it does not waste the blood from the system. Blisters and sinapisms may also give relief in slight cases; but they have little power where, as is commonly the case, the whole posterior parts of both lungs are congested with blood, perhaps in a morbid state. The principal remedy in this form of pneumonia (if pneumonia it can be properly called,) is mercury, which may be combined with opium, saline medicines, and even with stimulants, in certain cases; but these are points of practice too delicate to be detailed here.

* I understand that my originality in recommending the "dry treatment," in catarrhal colds, is disputed by M. Piorry, who states that he has successfully treated bronchitis by abstinence from fluids for six years past, and that he has described and recommended this mode of treatment in his lectures, and (I believe) in the *Bulletin de Clinique*. I have at present no means of referring to the precise date of M. Piorry's publication; but that I preceded him in the practice will be pretty evident when I state, that the article *Coryza*, in the *Cyclopædia of Practical Medicine*, in which the "dry method" is recommended for a recent cold, was written in 1831, and published in May 1832; and I had been in the habit of using it since 1826, and soon after that time recommended it to many medical friends in London and in Paris; among others, I may name Sir James Clark, Sir Benjamin Brodie, and Sir David Davies. I certainly never learnt the practice from any one, but others may have used it without my knowledge; and my friend, Mr. Keate, has lately informed me that his uncle did so many years ago.

The complication of pneumonia with bronchitis is generally a fit subject for blood-letting, followed by the antimonial treatment. It generally terminates by free expectoration ; and expectorant mixtures and blisters are of more use than in simple pneumonia. In pleuro-pneumonia, local as well as general blood-letting should be practised freely ; and after the more acute stage has subsided under the influence of these and of antimony, if the buzzing bronchophony and dulness on percussion still continue, the patient should be blistered and put under a mild course of mercury, to promote the removal of the fluid and interstitial lymph, which might lead to partial obliteration of the tissue of the lung.

We have hitherto said nothing of *chronic pneumonia* ; we must not, however, pass it over ; for although Laennec almost doubts its existence, at least as a separate affection, I by no means partake of this doubt. As we have seen, with regard to pleurisy, so in pneumonia, the inflammatory action may not entirely terminate with the effusion of lymph, although it do not lead to the third or suppurative stage. When the acute inflammation is extensive, and the effusion of lymph is not removed by absorption, the disease generally proves fatal before there is time for further change ; but in circumscribed peripneumonies, or in small parts of more extensive hepatization, a chronic inflammation sometimes goes on, and produces that kind of tough induration which is the general result of chronic inflammation in a parenchymatous structure. Thus, in the lungs of those who have suffered from long and repeated attacks of inflammation of the chest, even where there are no tubercles, we not unfrequently meet with portions of the tissue which are dense, almost destitute of air and of liquid, tough, and sometimes almost cartilaginous. Their colour varies from a dark dingy-red colour to different lighter shades of reddish brown and buff, sometimes rendered grey by a mixture of the black pulmonary matter. Their aspect also is varied like that of acute hepatization, by the tissues that are most affected, it being sometimes granular or oölitic (as Laennec has described it,) from the especial thickening of the individual vesicles ; in other cases streaked or veined, from the hypertrophy of the interlobular septa and cellular tissue under the pleura and around the large vessels ; in others more uniform, and of a darker colour, from the pulmonary plexus of the vessels being the chief seat of the alteration, and the colouring matter of the blood, entering largely into the deposition. In this last variety the cellular tissue between the lobules and under the pleura is somewhat thickened to the amount of several lines, and is of a light drab or grey colour, like that of miliary granulations, and like them has almost the density of cartilage. Now these changes, which thus occur as the sequel of acute pneumonia, are also frequently met with complicated with those states of the lung which are called tuberculous ; and a considerable portion of the consolidation that is met with in phthisical lungs, often presents precisely the same anatomical characters as these chronic hepatizations which supervene on acute pneu-

monia imperfectly subdued. I have also more than once met with them in the lungs of those who have long suffered from extensive organic disease of the heart, where the circulation through the lungs was perpetually impeded by the structural lesions of that organ ; and it seems to me that the same mechanical congestion from this cause, which sometimes leads to an effusion of blood in the tissue, constituting pulmonary apoplexy, in other cases, if long enough continued, terminates in an effusion of lymph, and an obliteration and consolidation of the pulmonary tissue. The condition of the lung is sometimes coupled with irregular dilatation of the air-cells ; and on examination, after death, the organ presents a knobby surface, and feels nodulated where the consolidation occupies parts or lobules. I think that there is reason to ascribe also to a minor extent of chronic inflammation of the parenchyma, that increased density and rigidity of the lung, without entire consolidation, which is often found in the lungs of those who have long suffered from dyspnœa.

The signs of chronic peripneumony are those of consolidation and obstruction of the vesicular tissue, which continue after the urgent symptoms of the acute disease have subsided. The dyspnœa has become less oppressive, although it is still felt on exertion ; the fever has been reduced, although there is still some quickness of pulse, and a return of heat of skin towards night ; there is still some cough ; and although there may be improvement in strength and appearance, it is not progressive ; the patient remaining with his organs and functions abridged ; there is still dulness on percussion, with bronchial respiration and vocal resonance in the seat of the late inflammation ; and if, in this condition, he neglect the means which may most conduce to the better restoration of his health, the indurated portions of the lung may either prove centres of fresh attacks of acute inflammation, or they may themselves spread, ulcerate, and commence the career of a phthisical disease, which sooner or later will destroy life. I have met with several cases of consumption that have appeared to originate in this manner, independently of any tuberculous disease, or diathesis ; the individual having been in excellent health, and quite free from all chest complaints before the attack of acute inflammation, which, afterwards degenerating into this chronic form, laid the foundation of a consumption which ultimately proved fatal. It is, however, much less untractable and slower in its progress than the true tuberculous consumption ; for the disease is more local than constitutional, and if circumstances do not occasion its extension, and injure the constitution by a constant and increasing inroad on the functions of respiration and circulation, it may be in the power of nature to effect its removal. I shall resume this subject in connexion with that of Phthisis, and shall merely remark here with regard to the treatment of chronic pneumonia, that in the first instance a mild course of mercury, with external counter-irritation, and afterwards the use of hydriodate of potash, with sarsaparilla, or some similar alterative, with mild sea air, regular

gentle exercise, and a well-regulated diet, have been, in my experience, the measures most deserving of recommendation.

I must now take a rapid glance of the other diseases, which are seated chiefly in the parenchyma of the lung, and are not essentially inflammatory.

Œdema of the lung is an effusion of serum into the parenchyma, probably both between the cells and minute tubes, and within them. Its anatomical characters are, that the lung is paler and heavier than usual, pits on pressure, and crepitates little under the finger. It is seldom idiopathic, but, like *œdema* of other parts, results from some loss of balance in the circulation, an obstruction to the return of blood, or occasionally an excess of exhalation. Thus it may arise from the obstructions occasioned by organic diseases of the heart, lungs, or liver; or from the increased exhalation supervening on exanthematous diseases, particularly scarlatina and rubeola; or on those diseases of the kidneys which interfere with their excretory function, and are accompanied by dropsical effusions in various parts.

Being thus a sequel of other disease rather than a distinct pathological condition in itself, it must vary greatly according to the cause which produces it. When extensive, it occasions dyspnoea, cough, and thin mucous or serous expectoration. The physical signs are a crepitating or *sub-crepitant* rhonchus with the breathing, less fine and even than that of pneumonia, and giving proof of the presence of more liquid by the mucous rhonchus in some of the larger tubes. The natural vesicular murmur is rendered indistinct, especially at the lower and back parts of the chest, where also the sound on percussion is somewhat impaired. Now you see these signs are very like those of the first stage of pneumonia; and although I have said there is some difference in the character of the crepitation, yet this is scarcely marked enough to constitute it a distinction; and a better source of diagnosis is in the general symptoms, the fever, rusty expectoration, and progressive increase of pneumonia being absent, and there being present *œdema* of other parts, or other signs of disease that may be supposed to produce it. In fact, I should say, from my own observation, that the lungs are less liable to *œdema* than the external cellular tissue, and that you scarcely ever have *œdema* of the lungs without anasarca, or at least partial dropsical effusions in the limbs.

I can say no more of the treatment of *œdema* of the lungs than I did of that of hydrothorax; it must depend on the nature of the cause. The *œdema* which follows scarlatina generally soon yields to hydragogue purgatives and digitalis.

Pulmonary hemorrhage, or *apoplexy of the lungs*, is an effusion of blood into the parenchyma and vesicular structure of the lung. The name *apoplexy* is by no means appropriate; for this word by etymology implies only the chief symptom which accompanies the analogous lesion of the brain, its striking-down or stunning effect, which by no means belongs to the lesion as it affects the lungs. The effusion of blood in the tissue of the lungs may arise

from three classes of causes:—1. From circumstances obstructing the passage of blood through the lungs, such as diseases of the heart, especially those impeding the passage of the blood through the left side of the heart, with hypertrophy of the right ventricle; tubercles or other deposits in the lungs, compressing the blood-vessels; violent acts of exertion. 2. From a diseased state of the pulmonary vessels, or parenchyma generally, so that any temporary congestion from exertion, or the continued exercise of the voice, occasions a rupture: in phthisis there is often both this fragile state of the vessels, and an obstructing pressure on them, and the same combination of circumstances produces pulmonary hemorrhage in some diseases of the heart and arteries. 3. A dissolved or scorbutic state of the blood is another cause to which I should also refer some cases of pulmonary hemorrhage; for I have several times, in fevers of a petechial kind, met with lungs presenting circumscribed portions of their tissue quite infiltrated with black blood, with effusions of the same kind under the pulmonary pleura, there being no signs of inflammation, and the adjoining tissue being free from disease.

The effect of these extravasations of blood is to produce patches more or less extensive of a very dark red or brownish red colour in the pulmonary tissue, their margins being often quite abrupt, as you see in these drawings. These patches are generally pretty solid, from the coagulation of the blood, and sometimes exhibit somewhat of a granular texture, like that of some kinds of hepatization; but the colours are all much darker. The hemorrhagic masses may vary in size, from that of a hazel-nut to the whole extent of a lobe of the lung. In the larger masses it is common to observe in the centre grumous or fluid blood, and there is obviously considerable destruction of the tissue. It is not uncommon to find purulent and gangrenous matter in these hemorrhagic portions; and this is not surprising, seeing that the effusion of blood not only breaks up and destroys the tissue in parts, but also, by its pressure, sometimes obstructs the vessels, and thus destroys the life of the parts which they supply. So, also, you may have hemorrhagic engorgement associated with pneumonia in any of its stages; for the local obstruction and irritation of a clot of effused blood may be sufficient to determine inflammation of the adjoining tissue; and the distension of the vessels in the first stage of pneumonia may also, in those predisposed to it, be sufficient to occasion pulmonary hemorrhage. The hemorrhagic or inflammatory engorgements are likewise sometimes associated together with those congestions which are produced by obstructions to the passage of blood through the heart; so that in the lungs of patients that have lingered for some time with organic disease of the heart, we not uncommonly find apoplectic or hemorrhagic spots, inflammatory congestion, and hepatization, occurring together.

The symptoms of pulmonary apoplexy or hemorrhage are none of them constant. There may be dyspnœa, a feeling of tightness or dull pain in the chest, sometimes referred to a particular spot; cough; and what is most common and characteristic, hemoptysis. The blood

expectorated may be only little, and merely tinging the sputa, or it may be coughed up as pure blood ; or it may be brought up in large quantities, often containing coagula, by an action like vomiting. The latter kind is generally associated with tuberculous disease, where a vessel of considerable size has been ruptured, and communicate with a bronchial tube. In the case of the hemorrhagic masses in the parenchyma of the lung, associated with disease of the heart, there is sometimes little or no hemoptysis, the effused blood coagulating in the tissue before it reaches the larger bronchi. The blood expectorated is generally, but not always, frothy and florid, and mixed with mucous sputa ; and it is thus distinguished from that vomited from the stomach, which is more constantly dark coloured and mixed with liquid or other contents of the stomach. I have, however, often seen quite dark coagula coughed up ; and when the hemorrhage subsides, the blood in the sputa acquires a brown tinge. So in examination after death we find the hemorrhagic spots of some standing, of a deep brownish red colour. Sometimes the expectoration of blood is followed by a relief to the dyspnœa and tightness of the chest ; in other instances it is accompanied by feelings of faintness, and where the quantity of blood is large, its effusion may cause extreme danger, and even death by syncope, or suffocation. Or if the immediate effects of loss of blood be borne, there may follow the jarring pulse, palpitation, anhelation, tinnitus aurium, and other symptoms of the reaction or irritation which ensues on excessive inanition of any kind.

The physical signs of the effusion of blood in the tissue of the lungs are such as you would expect from the filling up of some portions of the tissue, and the pressure on the parts adjacent. Thus there may be some part of the chest in which may be found dulness on percussion, with absence of vesicular respiration ; and if the consolidation be extensive, there may be bronchial respiration, and vocal resonance. Around this part, which may vary in extent, there may be an irregular crepitation, with a sound of bubbling in larger tubes, arising from the pressure of the coagulated blood on the adjoining texture, and the presence of some in a liquid state in the outer tubes and vesicles. When the disease is extensive, and especially if complicated with tubercles in the lungs, the expansion of the chest will be imperfect on one side. When the foregoing signs present themselves in addition to hemoptysis, they pretty clearly trace the hemorrhage to the lungs ; but if hemoptysis be absent, they can only afford a strong suspicion, in addition to the presence of the causes likely to produce it, that there is hemorrhage or apoplexy in the tissue of the lung. For the dulness on percussion, the bronchial respiration and crepitation, may be equally produced by partial pneumonia ; and if there be at the same time bronchitis, the glairy sputa may get a rusty tinge, like those of pneumonia, from the blood effused, without enough admixture of blood itself to constitute hemoptysis. These constitute difficulties in diagnosis, but the practice required is not equally perplexing.

Whenever some or all of the preceding signs are joined with fulness and strength of the pulse, it will be expedient to bleed and purge, according as the strength may bear, and further to reduce the arterial action by the use of digitalis, a spare diet, and a limitation to the use of cold liquids, and that in very sparing quantity. When hemoptysis is present, an internal styptic must be also administered; and the most efficacious of these is the superacetate of lead, which has really appeared to me in several instances to restrain the hemorrhage. It may be given in two or three grain doses, combined with the aqueous extract of opium, repeated every third hour or oftener, according to the urgency of the hemorrhage. There is one point to be especially attended to when the hemoptysis has been arrested, and there are still dulness on percussion and crepitation in some part of the chest; the infiltrated portion of tissue, or that adjacent to it, may become inflamed, and even destroyed by suppuration; and it is of great importance to check, by seasonable depletions, the tendency that is thus shown to a lesion, which under these circumstances may become destructive to the tissue of the lung. A free bronchial secretion, provided it be chiefly mucous and only tinged with blood, is not unfavourable; for it tends to relieve the pulmonary engorgement, and I have known signs of hemorrhagic consolidation of the lung after hemoptysis gradually disappear during the expectoration of a pretty copious glairy mucus, bloody at first, afterwards becoming brown, and finally opaque and muco-purulent.

I have thus given you a brief account of the diseases of the parenchyma of the lung which especially affect its vascular function, inflammation, œdema, and hemorrhage; it now remains for us to consider some changes in the vesicular structure, which alter the proportions of its parts, and impair its aptitude for the performance of its proper office, namely, *vesicular* and *interlobular emphysema*. These lesions, although they have some resemblance to each other in their name and causes, differ essentially in their anatomical characters.

Vesicular emphysema is a dilatation of the air-cells of the lungs. It may be partial or general; and the partial kind may be confined to separate vesicles, or it may affect all the vesicles of a lobule; general dilatation may affect one lung, or the whole, or a considerable part of both lungs. The anatomical characters of these lesions present some variety, but they have this in common, that when examined either through the pleura in the recent state, or after being inflated, dried, and sliced, the air-cells are seen much larger than those of healthy lungs. When the dilatation is general, the pleural surface of the lungs may be as smooth as usual, only more convex; but, when it is partial, you see either the dilated vesicles or the emphysematous lobules, forming irregular prominences. Individual vesicles are sometimes seen under the pleura, and especially at the margin of the lobes, dilated to the size of a pea, a hazelnut, and in some cases to a much larger size.

I have noticed another remarkable difference between cases of

dilated air-cells, whether general or partial. Some are accompanied with a diminished pliability of the texture of the lung ; it has acquired a degree of rigidity ; it does not collapse when the chest is opened, and gives a greater resistance to the fingers than a healthy lung does ; this kind is generally accompanied by a great deal of the black pulmonary matter. There is here, notwithstanding the dilatation of the air-cells and lightness of the lung, a hypertrophy and toughness of some of its textures ; and portions near the root are sometimes found approaching to the dry tough consolidation of chronic pneumonia, but still containing a good deal of air.

The condition of the lung in the other cases is just the opposite of this : the texture is more flaccid and yielding than usual ; and when the margin of a lobe is pressed between the finger and thumb, it feels almost like a soft single membrane, it is so thin. It often does not collapse on opening the chest, and this appears to be from a loss of elasticity, for it pits on pressure, like an œdematous lung, and does not recover its shape again, and like it, too, it is commonly much paler than usual. Andral has noticed this condition of the lung under the name of *atrophy*, and observes that it occurs most commonly in old people. I have seen it both general and partial ; the partial kind of flaccid dilatation being of common occurrence in the anterior lobules and margins of the lungs in tuberculous phthisis. The fringe of dilated cells, like a row of beads, which is sometimes seen at the margin of the anterior lobes, is also simple dilatation without the least rigidity. On examining the dilated cells, they are generally seen to be smooth, and lined with membrane within, but I have several times found them communicating with one another ; so that, on inflating one, the air entered those adjoining. They have been sometimes found to contain serum, pus, and even tuberculous matter. These several lesions are frequently associated with diseased conditions of the bronchial tubes, redness and thickening of the mucous membrane, hypertrophy of the longitudinal fibres, dilatation, and partial contraction.

We shall better understand the pathology and signs of these affections, if we inquire a little into the manner in which they may arise from the lesions which are known to precede and accompany them. You remember the account which I gave of the production of dilatation of the bronchi, and you will find the same views applicable to the dilatation of the air-cells. Laennec explained the origin of this lesion in this manner :—In cases of chronic catarrh, particularly of the dry kind, the small bronchial ramifications became so obstructed by the swelling of their membrane, or by the secretion of a viscid mucus, that the air can be forced through them into the vesicles only by an effort. Now, as in ordinary respiration, the inspiration, a muscular effort, is more forcible than expiration, the former may prove sufficient to overcome the obstacle to the introduction of air into the vesicles, while the expiration is inadequate to effect its expulsion. Successive portions of air, expanding by the increased temperature, are thus introduced and incarcerated in the

cells, which are thereby kept in a state of continual dilatation. This may be one mode in which the air-cells become dilated, but I think that we shall find other causes still more efficient and common in operation. When there is partial or complete obstruction in any of the bronchial tubes or cells, the inspired air cannot press with the usual force beyond the obstructions ; but it will press with more than usual force into the adjoining tubes and cells, to which its access is quite free, and these latter may thus become distended, and in time permanently dilated. The obstructions to which I allude may be caused by the viscid secretions in the tubes, thickening of the textures, tubercular deposits, and the like ; and it is with diseases in which these occur, that dilatation of the air-cells is most commonly associated. Tuberculous or other solid deposits obstructing small parts of the vesicular texture chiefly, are especially calculated to cause the dilatation of the cells immediately adjoining ; for, rendering inextensible the parts which they occupy, they cause the force of full inspirations to fall on the pervious tissue ; and we accordingly find these deposits almost constantly accompanied by pulmonary emphysema, which makes prominent the lobules or parts of lobules on the surface of the lung after death. Another cause of dilatation of the air-cells, and this of a pretty extensive kind, is rigidity, or want of extensibility of the longitudinal fibres of the bronchi. This change I have already noticed as an effect of chronic bronchitis, and you can readily perceive that if the tubes do not lengthen with the expansion of the chest, the air will press unduly on the peripheral cells, and occasion their dilatation. Thus we find those at the margins of the lower lobes most dilated, for these are most under the influence of the forces expanding the chest. It is not uncommon to find the marginal vesicles dilated in the lungs of old people with ossified cartilages ; and I think we must ascribe this also to the comparatively immobile state of the ribs and central portions of the lungs, and the increased action of the diaphragm, and consequent undue pressure of the air into the texture immediately contiguous to this part of the respiratory apparatus. These are the chief mechanical causes by which the vesicular texture of the lung becomes distended ; but there are others of a more vital nature, which may also be concerned in the production and perpetuation of this lesion.

M. Andral conceives that the air-cells sometimes become enlarged by a wasting away and breaking down of some of their walls, so that several are reduced to a few of larger size. This may be, doubtless, a cause of the unusually light and flaccid condition of the lungs often found in old people after death ; but such a state simply would not constitute the disease which, during life, is so cognizable as vesicular emphysema. I have before said that an unusual flaccidity often attends dilatation of the air-cells, and this probably proceeds sometimes from atrophy, and sometimes from a loss of the proper elasticity of the textures. The analysis which I have given you of the structure and properties of the air-tubes and cells will suggest the existence of another cause of dilatation, in a defective tone of the

circular fibres, as well as in a loss of elasticity in the longitudinal. I formerly mentioned to you the fact observed by Mr. Swan, that in animals which died after the eighth pair of nerves had been divided in the neck, the lungs were found uncommonly distended with air. Again, it was a remark of Laennec, that the lungs of persons who had been asphyxiated by the air of sewers, were always very voluminous, yet filled with air, and he proposes a question whether this may be an idiopathic dilatation of the air-cells. In fact, if the contraction of the circular fibres, be an essential part of natural expiration, it is plain that such influences as those just mentioned, which weaken or destroy their power, must render expiration imperfect. Dr. W. Stokes, of Dublin, has taken this view of the subject, and supposes that paralysis of the bronchial muscles, is the chief cause of dilatation of the air-cells. Following the same view which Dr. Abercromby had advanced with regard to the intestinal muscles, he considers inflammation of the tunics investing the tubes, such as that of bronchitis, to be the cause of this paralysis. I cannot, however, consider the defective action of the bronchial fibres as more than an aiding cause in the production of pulmonary emphysema; it may do more in determining dilatation of the tubes; but the contractile power of the vesicular tissue is not clearly proved, and I think that the mechanical causes to which we have been adverting must be viewed as the chief agents in distending this tissue, which is naturally so yielding and ready to adapt itself to a change of pressure.

Reverting to the anatomical differences of the *tense* and the *flaccid* vesicular emphysema, we may as well trace in one the effect of an over active and irregular nutrition of the textures, the common result of repeated or prolonged inflammation: and in the other, the absence of any such process, if not the presence of one of an opposite character, causing a wasting of the same parts. Now you must perceive that these opposite conditions will lead to very different effects in the signs and course of the lesion. In the one case, the lungs become comparatively fixed in a distended state; and as they resist the power of the expiratory forces to expel the air from them, unusual exertion is required in inspiration to introduce enough air to serve the purpose of respiration. They are, therefore, perpetually exposed to a distending force; and as the dilation proceeds, and the increasing rigidity of obstruction with it, the lungs acquire a permanent volume beyond what is usual, even in full inspiration, and they distend the walls of the chest, and press on and even displace the adjoining organs and vessels. Hence may be expected to arise continual oppression of the functions of both respiration and circulation, and cachectic and dropsical disease in the system as the sequel. In flaccid vesicular emphysema, on the other hand, there is little or no increase of volume of the whole lung, and no pressure on the other contents of the chest. Having arisen from the access of air to the vesicles being more free than the vesicles themselves were capacious, there is not that tendency to accumulation which accompanies the emphysema arising from inflammatory thickening or injured motory power in the tubes.

In fact, in flaccid emphysema the tubes are often also dilated, and afford pretty free egress as well as ingress to the air. But the air thus admitted to few dilated cells cannot aerate the blood as it would in many small cells; it is out of proportion to the vessels and blood in the part. Hence, however this kind of emphysema may be caused, it will not improve the function of the lung, although it supplies more air to it.

I think you will now understand what we have to describe of the symptoms and signs of vesicular emphysema. Inasmuch it is permanent, it will cause a constant shortness of breath, or dyspnœa; and the least additional obstruction, such as that of a cold, bodily exertion, or flatulent distension of the stomach, may increase this symptom to an oppressive degree; so as to resemble an attack of asthma. The permanency of the oppression to the function of respiration, in severe cases, induces a cachectic state of body, which is manifested by palidity and some emaciation, and a depraved condition of all the excretions. In the tense form of emphysema, with increased volume of the lungs, there are added the symptoms of obstructed circulation, as well as imperfect oxygenation of the blood, lividity, and even blueness of the face and lips, dropsical effusions, palpitation, and other signs of hypertrophy of the heart. Dr. Stokes has remarked, that these symptoms are always worse in cases where the lower lobes of the lungs are chiefly affected, which he explains by the greater injury to respiration, which would be caused by the enlarged lung preventing the free play of the diaphragm. The expectoration accompanying emphysema is very various. It is most commonly mixed, a dirty serous fluid, containing portions of tough pearly mucus, or of the opaque sputa of chronic bronchitis. In the attacks of acute bronchitis which frequently occur, it becomes glairy, and often very copious towards their termination, like in pituitous catarrh.

Of the physical signs of pulmonary emphysema, one of the most remarkable is the loud hollow sound on percussion, which is even greater than that of a healthy chest. This is common to both the tense and flaccid kinds of emphysema; but in the former, when extensive, there is a distinctly raised pitch in the sound of percussion, such as in a less degree you can produce by striking the chest when you hold in a very full breath. As in this case, in fact, the walls of the chest are rendered more tense by the increased volume of the lung, and the vibrations which they make are therefore quicker, although from the elasticity of the contained material, they are still quite free. The increased volume of the lung is manifest in advanced cases, in the shape of the chest, which is unusually convex or rounded. The sides, the front, the back, and even the supra-clavicular spaces, some, or all, present this rounded projection; and according to Dr. Stokes, when the lower lobes are affected, the heart, the liver, and the spleen, may be displaced by the emphysematous lung, which then yields its clear resonance on percussion over an extended region. In this tense kind of vesicular dilatation, the sound of respiration is

very imperfect and wheezing, and forms a remarkable contrast to the efforts used to introduce and expel the air. This is perceptible to the eye as well ; for it can be seen that, with all the exertions in expiration, the chest is very little diminished, and retains its large convex shape, whilst every intercostal, and every supplementary muscle, can be seen at work, endeavoring vainly to depress the ribs. Is there any wonder that this force, continually exerted on the thoracic vessels through the stuffed lungs, should obstruct the circulation, and cause lividity, cyanosis, dropsical effusion, and ultimately disease of the heart? Inspiration is easier, but even this requires exertion ; for breath has to be taken, as it were, on the top of breath, and even needs the supplementary effort of the cervical and superior dorsal muscles. There are sometimes heard some odd sounds, which are not those of common wheezing or impeded breathing. Now and then there is a sudden clicking or crackling, as if from the sudden passage of air into, or out of, a set of tubes and cells which were before closed. Sometimes there is a sound of friction, like that of a finger rubbed on a table, which may, perhaps, be produced by the rubbing of projecting lobules or cells against the costal pleura.

The signs of the flaccid form of emphysema may be much modified by the other disease, such as tuberculous deposit, that commonly produces it, but they are essentially distinct from those of tense emphysema ; and neither this nor their anatomical difference has been, so far as I know, noticed by authors. There is the clear sound on percussion, but no raising of the pitch of the sound. In old people, where this form of emphysema is uncomplicated with solid deposit, the sound on percussion is clearer and deeper than in any other case. Neither the shape nor the motion of the chest is materially affected by this kind of emphysema ; and the sounds of respiration, instead of being obscure and wheezing, are remarkably loud, and even puerile, although they have sometimes a little of the whiffing or bronchial character, from the accompanying dilatation of the tubes. You will understand the reason of all this when you reflect that, in these cases, air passes freely into, and out of, the dilated cells, and that their enlarged size, together with the increased energy which the feeling of dyspnœa gives to the acts of respiration, is a sufficient reason why the sound of respiration should be louder than usual.

I have little to say about interlobular emphysema. It is essentially distinct from the preceding affection, although it may be combined with it. It is an effusion of air into the cellular membrane of the interstitial tissues of the lung, and therefore especially between the lobules and under the pleura. You see by these drawings that it is easily distinguished anatomically, by the air being in the line of the interlobular septa, and contained in angular cells of various shapes and sizes, and not round ones, like those of the lung. Sometimes air is effused under the pulmonary pleura, detaching it from the lung in the form of large bubbles. This affection is commonly produced by violent efforts, or by wounds of the lung ; but sometimes from rupture of the air-cells by excessive or sudden dilatation. It may, if

extreme, produce sudden, and even fatal, oppression to the breathing ; but in slighter cases it is of no consequence, being removed spontaneously. The only sign supposed to mark this affection is a sound of rubbing with the motions of respiration, which the projecting emphysematous septa make against the walls of the chest. This is sometimes in successive jerks, so as to resemble the steps of a person mounting and descending a ladder.

I have no time to say much on the treatment of emphysema. The prevention of it is the most attainable object ; and with this view, the removal of those inflammations which lead to an obstructed state of expiration, and the dispersion of the obstructions themselves, are the chief indications. The use of counter-irritants, alkaline attenuants, and other remedies recommended for chronic bronchitis and dry catarrh, is the most likely to fulfil them. Where there is already evidence of loss of contractility of the pulmonary tissue, mildly stimulating expectorants, and especially inhalations, may be of some avail ; and frictions of a strongly exciting kind to the exterior of the chest, or blisters, are certainly of some benefit. Dr. Stokes mentions strychnia as likely to restore, in some measure, the lost contractility of the circular fibres. The symptoms which arise in inveterate cases of pulmonary emphysema must be treated on general principles, remembering that although we cannot remove the cause, we may somewhat prevent its increase, and diminish its aggravations from temporary circumstances. The flaccid form of emphysema is scarcely a matter for treatment : we cannot increase the number of the pulmonary cells, but we may in some degree so regulate the body as to diminish the want of breath, and make the small number suffice. With this view, a tranquilizing plan of medicine and regimen, avoiding all circumstances which may tend to excite the circulation or respiration, at the same time promoting the due activity of the secretions and tone of the system, by gentle exercise and alterative tonics, may serve to keep up a tolerable balance of imperfect health, and prolong existence on a lower scale.

LECTURE XXI.

Diseases of the Parenchyma of the Lung (continued).—Phthisis Pulmonalis.—Anatomical History.—Granulations.—Diffused Induration.—Yellow Tubercle.—Tuberculous Infiltration.—Vomica and Cavities.—Pathological History.—Nature of the Indurations ; Views of Laennec, Andral, and Carswell ; Explanation of their Production and Changes.—Nature of Yellow Tubercle ; Views of Authors.—Explanation of its Production and Changes.—Causes of the Development of Phthisical Lesions.—Seat of Tuberculous Deposits.—Contraction and Obliteration of Tuberculous Cavities.—Cretaceous Tubercles.

THE last class of diseases of the lungs which I shall consider, are those comprehended under the head *phthisis pulmonalis*, or *pulmonary consumption*. By this name, and more popularly by that

of *decline*, is implied a wasting of the body from the effect of a disorganizing process going on in the lungs. It is, unfortunately, too familiar to us all, to need any further definition or description : and in order to preserve that rational connexion which these lectures are designed to exhibit, between the physical changes and pathological lesions, and the signs and symptoms which they produce, it will be well to give you, in the first instance, a brief sketch of the anatomical characters of consumptive disease of the lungs ; we shall then be able to trace it in its progress through its various stages and complications, and to comprehend the changes of physical properties which it may induce in the organs of the chest.

When we examine the lungs of persons who have died of consumption, we find them greatly changed from their natural condition: they are more or less consolidated in irregular masses, and on cutting into them they are generally also excavated in parts into hollows of various sizes, which are either empty or contain a thick liquid matter. On closer examination, and after attentively observing the lungs of many consumptive persons, we are enabled to classify the morbid conditions which they present under the following heads :—

1. On pressing the softer parts of the lung between the fingers, we feel in it a number of hard little bodies, and on cutting into it we see them roundish granules, of a light semitransparent reddish drab or skin colour, sometimes more grey or ash-coloured, more rarely devoid of colour, and quite transparent, of sizes varying from a pin's head to a hemp-seed. Their hardness is considerable, sometimes almost equalling that of cartilage ; these are the *miliary granulations* or *miliary tubercles* of Laennec and other writers. They are sometimes found singly, studding a tissue otherwise healthy ; but more commonly they are in groups of several together, and then they are either clustered in bunches, like berries, or they form a considerable mass ; with the interstitial tissue consolidated and indurated between them. They are most commonly distinct in the inferior lobes ; in the upper parts, and near the root of the lung, they are usually conglomerated in masses. In the upper parts, too, it is common to find in them opaque specks of a yellowish white colour, which are generally in the centres of the granules, sometimes at their margins. —In the distinct granulations the opaque part is little more than a speck ; but in those which form a conglomerated mass, the opacity is sometimes seen extending from granule to granule ; and in others it constitutes a mass of considerable size within the mass of granulations.

2. In the next place we find consolidation of another kind. It is diffused through some extent of the pulmonary tissue, of no particular shape, except that sometimes it seems to be limited to single lobules. In consistence it varies, but often it is nearly as hard as the miliary granulations, and in parts it has somewhat of their semi-transparency and colour, but generally it has a darker hue, from the colour of the blood and of the black pulmonary matter in it. The consolidation is pretty complete, and the pulmonary texture cannot be dis-

tinguished in it, except here and there the coats of a large blood-vessel, bronchus, or an interlobular septum, which are often thickened, and partake of the induration. In other cases the consolidation is less perfect, there being still some air in the tissue, and the adjoining tissue being often emphysematous. In these indurated masses are often to be seen, here and there, more opaque lighter-coloured spots, which are sometimes quite distinct and of a dead yellowish-white, like those seen in the miliary granulations; but they are here less regular in their shape and size, being sometimes in streaks, curves, and angles, and mottling the dark consolidated texture with spots and patches of a lighter and opaque hue. In the lightest and most opaque spots we recognize what we must describe as the third morbid appearance to be met with in phthisical lungs, namely:—

3. Opaque yellowish-white masses of various form and size, generally somewhat rounded. Some of them are nearly as solid as the dark or semi-transparent indurations, but they are much less tough; others have more or less of a cheesy consistence, and some are found in parts approaching to a state of grumous fluidity, still retaining their light colour and opacity. These opaque masses are commonly found within the indurations from which they appear to be formed, and they are just of the same character as the specks before described as occurring in some of the single or aggregated miliary granulations.—In fact, as these specks are seen in some in greater number and extent, and preceded by an intermediate state of opacity, in parts to pervade the whole mass, it may be fairly concluded that the clusters and nodules of granulations are also converted into this same opaque, friable, yellowish-white matter. This matter, which is indisputably entitled to be distinguished as *tuberculous*, is occasionally found also in other situations, unaccompanied by any induration; such as in the interior of dilated vesicles and bronchial tubes, in masses under the pulmonary pleura, and in the bronchial glands. In these instances it is commonly of a friable or cheesy consistence, and has not the hardness which it seems to retain for a while when it has originated in the indurated tissue. But this yellow tuberculous matter, however tough and hard it may be in the first instance, tends to soften, either partially or wholly; and thus the masses are sometimes found consisting of loose clots, in a purilaginous fluid, or wholly reduced into a curdy kind of puriform matter. The tuberculous matter is also not unfrequently found diffused through a considerable extent of the pulmonary texture, constituting the *infiltrated tubercle* of Laennec. In its earlier condition, the lung, in this state, closely resembles the last stage of hepatization, when the opacity, which precedes suppuration, shews itself. It is very much mottled or marbled, for besides the yellowish-white opacity which is seen in different degrees in its different parts, there is the black pulmonary matter giving it a grey or greenish colour; there are the whiter coats of vessels and interlobular septa, and spots of red tissue less affected.—When the lung in this state is cut or torn, which it commonly may be with facility, its interior presents a granular surface, like that of

hepatization, and except that its colour is more varied, and it has generally more of the light opacity of tuberculous matter, it resembles a hepatized lung more nearly than any thing else. But in it are seen what are rarely met with in hepatized lungs, circumscribed abscesses or cavities, containing a fluid matter. To this softened and fluid state, then, all the conditions that I have been describing, tend to pass; and they constitute *vomicæ*: when so softened, the matter is evacuated into the bronchial tubes, leaving behind the lesions, which I have next to describe.

4. Lastly, we find cavities or excavations very various in number and form, and of sizes from that of a cherry-stone upwards, to the extent of a whole lobe. Sometimes they contain more or less of the remains of the softened tubercle, or a more liquid pus, or a mixed serous or mucous fluid tinged with blood, or they may be empty.—They communicate with each other and with the bronchial tubes, the process of softening and ulceration having destroyed the terminations of these tubes; but blood-vessels and interlobular septa are often spared by the destructive process, and form cords or bands across the cavities. The blood-vessels are, however, almost always impervious in these cases, and the septa are thickened by depositions of lymph. Their walls are composed of the consolidated tissue of the lung, rough, and occasionally sloughy, or of an irregular coat of lymph, or in old cavities of a sort of adventitious membrane, which is sometimes thin, like a mucous membrane, and in some more rigid, and of a fibro-cartilaginous character. When these cavities approach to the pleural surface of the lung there is often a coating of lymph or false membrane on the pleura at the part, which either thickens it, or unites it by adhesions to the costal pleura. Sometimes, however, there is no such deposit or adhesion; and it occasionally happens that the pleura is also ulcerated, gives way, and being perforated, allows the contents of the cavity, and the air from the bronchi, to pass into the pleural sac, constituting pneumothorax and pleuritic inflammation. There is this remarkable in the position and size of the cavities, that they are almost always largest and most numerous near the summits of the lungs, there being often one or more cavities there, when in the inferior lobes there are only scattered indurations. In fact, it may be generally observed of all those lesions connected with phthisis, that they affect the upper and posterior more than the lower and anterior lobes; and that they are also more advanced in the former. This is, however, most remarkable with the circumscribed indurations and tubercles; for with the diffused consolidations, especially of the light opaque kind (tuberculous infiltration), the middle and inferior lobes are often also affected, and cavities are more commonly found in every part.

Besides these chief and more essential changes of the lungs, in phthisis, many others are often found of a more accidental character, such as hemorrhagic effusion and consolidation, inflammatory congestion, and hepatization of the lung—products of inflammation in the pleura; inflammation, ulceration, thickening, and dilatation of the

bronchial tubes ; irregular dilatation of the air cells, sometimes with increased flaccidity, sometimes with rigidity ; enlargement and induration of the bronchial glands, with yellow tuberculous matter in its different states in them.

Let us now generalize a little on these changes, which anatomy has discovered to us in the lungs of the consumptive. They may, for the most part, be reduced to two. 1. Consolidation, generally of an indurated kind, and either almost colourless and transparent, or of pearly grey or reddish drab, or of a dark red or more dingy colour. 2. An opaque yellowish white, parsnip-coloured, friable matter, of various degrees of consistency, being first hard, and afterwards becoming soft, and forming vomicæ: this lighter opaque matter, which is properly called tuberculous, is produced commonly within the consolidations just named, but sometimes elsewhere.

What, then, are these morbid conditions of the lung ? What pathological view are we to take of them ; that is, what are we to think of their nature and origin ? Let us see first what the most eminent modern pathologists say respecting them. Laennec considered them "accidental productions, that is, real foreign bodies which spring up in the substance of the lungs, and may be developed in any other texture of the body." We owe great respect to the name of Laennec, for, as you know, we have largely profited by his labours ; but I must in candour confess, that his view of the origin and nature of tubercle has never been satisfactory or even intelligible to me. The only way in which I can interpret it is, that tubercles are parasitical bodies originating in an unknown way, possessing a life and structure of their own, growing by attracting matter to them, and tending, by their own inherent properties, to go through a certain series of changes. The transparent miliary granulations, the grey miliary tubercles, the grey diffused induration, and a gelatinous infiltration, he looked on all as varieties of these bodies in their first stage, and as all tending *per se* first to become opaque and yellow, then crude tubercle, which is still hard, and ultimately to soften into a cheesy or pasty liquid, which is the mature tubercle.

Now this view involves several assumptions little supported by analogy ; for instance, that bodies so different in physical character and texture are the same, and that the stages through which they pass are produced by their inherent properties, and not by the modified properties of the tissue of the organ ; and it assumes what has been disproved by observation, that the opaque yellow tubercle is always preceded by the grey or semi-transparent, and that the grey induration must always in time become yellow tubercle. Whilst, then we admit the accuracy of Laennec's observation, that the grey and semi-transparent indurations tend generally to become yellow tubercle, we must consider his view of the change to be too unsupported and hypothetical to be received as satisfactory.

The view of M. Andral is far more simple, and involves fewer assumptions. He considers tubercles generally to be the result of a

modified nutrition of the texture, and that they are produced, and go through their changes, through the agency of the vessels of the part and the blood which circulates in them. Although he admits that the miliary and diffused indurations precede the production of yellow tuberculous matter, he supposes them to be, not an early stage of this matter, but the result of chronic inflammation affecting the individual vesicles, or the general texture. The chief peculiarity of this view is, the explanation of the regular form and size of miliary tubercles, by locating them in the individual air-vesicles, just as the same author first accounted for the granulations of a hepatized lung. That the diffused induration, called by Laennec the first stage of tubercle, is the result of chronic inflammation, has been admitted by Chomel and Louis, who otherwise rather incline to Laennec's opinions.

The most recent writer on this subject is Dr. Carswell, with whose admirable "Illustrations of the Elementary Forms of Disease," I dare say you are familiar. He neither adopts the opinion that the indurations are an early stage of yellow tubercle, nor does he admit that they are more than accidentally connected with it. He supposes yellow tubercle to be a peculiar secretion, which takes place especially from mucous membranes, but that it may accompany other secretions, such as that of inspissated mucus in the air-vesicles, or of dense false membranes on the pleura or peritoneum; and thus he accounts for the grey miliary bodies with specks of yellow tuberculous matter, and the similar admixture of this matter with deposits on serous membranes. But if you examine the miliary bodies minutely, you will be soon satisfied that they contain no inspissated mucus that will account for their increased hardness; the induration is obviously in the texture itself, and not merely contained within the cells: and Dr. Carswell's view throws no light on the manner in which yellow tubercle is produced in the grey or dark indurations, whether miliary or diffused; yet this is a point as well established as any in the pathology of phthisis. I must not, however, take up your time by special pleading against particular opinions: let us examine the subject of the pathological changes rationally, in connexion with what we have learnt of the pathology of other textures, and we may come to a more satisfactory view than any which we have noticed. I must start by telling you, that I owe my notions on the matter, in a great measure, to Professor Alison; and although I do not know that our views are now quite alike, yet if I have fallen into the right track, it was his researches that directed me to it in the first instance.

If we examine the induration which commonly precedes the production of yellow tubercle, we find that it differs from the healthy structure certainly in these respects; that it contains a greatly increased quantity of matter, and that this matter is generally harder than the healthy tissue. Now this increase of substance implies either increased secretion or diminished absorption. That absorption is not diminished in the tissue, is plain from the fact that portions of the healthy tissue are at the same time removed by this process;

and that increased secretion is present, is proved by the fact, that the indurated texture presents new characters, and is not a simple accumulation of the matter of the natural tissue. Now to produce an increase in the nutritive secretion, there must, according to a well-established pathological law, be an increased determination of blood to the part. Does this amount to inflammation? Let us see whether indubitable inflammation ever produces an effect on a simple membrane, like that in question. In treating of pleurisy we found that acute inflammation of the pleura causes an overflow of the nutritive secretion in the form of coagulable lymph, which may soon become highly organised into a soft cellular membrane; but when the inflammation is of a lower and more chronic character, the effused matter is slower in the process of organization, and forms a harder texture of lower vitality—a kind of fibrous or fibro-cartilaginous structure. Will not the same observation apply to the parenchyma of the lung? The overflow of the nutritive secretion caused by acute pneumonia, we have found to constitute red hepatization, whether granular or diffused; but when we came to examine the effect of lower and more prolonged inflammation on the tissue of the lung, we had to describe a dark consolidation with increased density, in no essential particular differing from some forms of the indurations which we are now considering. Thus the hard, compact granular consolidation occurring around excavations, gangrenous as well as tuberculous, and admitted even by Laennec to be the result of chronic inflammation, has occasionally the colour and consistence of the indurations which precede the formation of yellow tubercle; and as I have shown that there is a non-granular form of acute hepatization, so it is reasonable to expect that there may be a diffused or uniform kind of consolidation resulting from chronic inflammation, affecting the interstitial more than the vesicular texture. To such a condition the grey diffused induration, called by Laennec the first stage of tubercle, so exactly answers, that Andral, Chomel, Louis, and Carswell, all concur in considering it to be a chronic form of hepatization. When it is the sequel of the acute disease, or of long continued pulmonary congestion, there is often much redness in the induration; but where the irritation has been of long continuance, and unattended with the more sthenic degrees of vascular action, or a very congested state of the lung, the texture is more semi-transparent, dense and grey, or variously modified by the black pulmonary matter in it. The more uniform colourless masses occasionally present, may be traced to the interlobular septa, or cellular tissue around the vessels, in a state of indurated hypertrophy. In these bloodless and almost cartilaginous portions, we see the exact characters of the matter of which the miliary granulations, or grey miliary tubercles, are minute samples; and if we adopt the view of Andral, that the regular size of these depends on the chronic induration being located in the coats of individual vesicles, we shall see a sufficient reason for their isolated or clustered characters. Moreover, as we have traced the diffused consolidations of the lung through various gradations, from acute,

soft, red, hepatization, down to grey induration, so M. Andral has found the miliary bodies presenting the same series of gradation ; being sometimes soft and red, in other cases livid and harder, whilst the same lung may contain also the granulations similar in size, but pale or grey, and of various degrees of induration. With regard to the more rare transparent miliary granulations of Bayle, described by Laennec also as an early stage of tubercle, I can state that I have twice, at least, found them on the pleura and on the peritoneum, when other parts of these membranes were coated with dense false membranes, and when there was no trace of yellow tubercle in the body. As to the commoner pale granular deposits on the serous membranes, they are the acknowledged products of chronic inflammation and their numbers and isolated circumscribed form constitute another point of resemblance to the miliary indurations which, in the lung, pass into the state of yellow tubercle. Without, then, going so far as to assert that the miliary indurations of the pulmonary tissue are always dependent on chronic inflammation, I think we may fairly say that both they and the diffused induration are more akin to the products of this process than to any other that we are acquainted with. The condition of the blood we found to be a material element in determining the products of inflammation in the case of pleurisy : so doubtless it is likewise concerned in the modified nutritive secretions of other textures. The more vital and organizable products are furnished by blood rich in fibrin ; and they are easily reabsorbed, or, if organised, are mobile, and sufficiently like the tissues of the part, not to incommode or irritate them. But if the blood be poor in nutrient matter, the deposit from it may be susceptible of only a low degree of organization, and will consequently be not only more difficult of absorption, but also less assimilable to the texture of the part, and more calculated to irritate it as a foreign body. It may thus be seen, that although the lowest degrees of inflammation may be alone capable of producing the chronic indurations, when the blood is healthy, yet, when it is diseased various degrees of inflammation, nay, even the ordinary nutrient process without inflammation, may be accompanied by the deposition of a lymph of a degraded character, and organizable only into a dense semi-cartilaginous tissue. I shall speak of this again presently.

The semi-transparent grey or dark induration is not always converted into crude yellow tubercle. Sometimes it is the seat of vomicæ, which contain a dirty or bloody pus ; and although even in this the curdy matter of tubercle is sometimes seen, it is plain that these vomicæ result from a more direct and speedy process of ulceration or irregular suppuration ; another analagous result of continued irritation in the condensed tissue.

And now let us pursue the same mode of inquiry with regard to the opaque pale yellow tuberculous matter which characterises the second class of phthisical lesions. Laennec calls this the second stage of tubercle, but neither for its formation nor for its subsequent softening does he assign any other cause than an assumed and unintelli-

gible "inherent property." Dr. Carswell is much more explicit on this point: in fact all his descriptions of tubercle apply only to this kind of matter. He considers it to be a secretion, *sui generis*, totally destitute of organization—an effete matter, continually separated from blood in an unhealthy state, thrown out chiefly on the free surface of mucous membranes, and producing bad consequences only in proportion as it accumulates in organs, impedes their functions, and acts on them as foreign matter. This opinion, so far as it regards the nature of tuberculous matter, does not differ materially from that long since published by M. Andral, who regards tuberculous matter as a peculiar modification of secretion, more analogous to pus than to any other matter; but materially differing from it. And truly, if we survey the general characters of tuberculous matter, consisting of pale, opaque, albuminous particles, generally deposited in a tissue previously consolidated, and the manner in which it tends to become soft and liquid, forming circumscribed collections like abscesses, or infiltrated through the texture, from which it is expelled like foreign matter, we cannot fail to see some general resemblances to the process of suppuration. We have found that the consolidating lymph of a hepatized lung becomes opaque and light coloured before it softens into pus; but the changes here are too rapid to admit of their being fully watched. But when an analogous process goes on more slowly, and in a simple structure, as in the pleura, we can better trace the resemblance. Thus in the latent and more protracted forms of pleurisy, we have had occasion to remark that the lymph first effused forms a dense tissue of low vitality, and resembling fibro-cartilage in hardness and colour. If the irritation still continues, this new structure throws out a lymph of still lower vitality, in friable shreds, and, in some instances, in form of a curdy matter, totally incapable of organization, which, mixing with the effused serum, constitutes one kind of empyema. Now such a process in the pulmonary tissue would produce all the changes which we have been describing, in the production successively of grey induration, crude tubercle, and softened tubercle. Thus a portion of this tissue (whether a single vesicle or part of a lobe), generally under the influence of chronic inflammation or local congestion, becomes indurated by the effusion of lymph susceptible of a low degree of organization. The original irritation continuing or the very induration itself determining an increased flow of blood to the part, the new structure evolves in the looser parts of its substance a still less organic form of albuminous matter, a pale opaque curdy substance; but as this cannot be (like that from the pleura) thrown off, it presses on its indurated matrix, and causing its absorption, accumulates in its place: thus is effected the conversion of the grey induration into crude tubercle. This entirely unorganized substance acting as a foreign body on the adjoining tissues and the remains of the living texture within it causes irritation, ulceration, and the effusion of serum and pus, which, as my friend M. Lombard, of Geneva, first explained, softens and breaks up the crude tubercle into the curdy

grumous matter of the matured tubercle. The same irritation and ulceration gives vent to the matter through one or more bronchial tubes; and thus are formed the tuberculous cavities.

But we have seen that yellow tuberculous matter is produced not only in the grey indurations, whether granular or diffused, but also in softer consolidations, like that of hepatization. It is sometimes seen in rounded circumscribed masses in a hepatised lung; in other instances it pervades with its opaque yellowish grey or mottled colour a whole consolidated lobe. In this, the infiltrated tubercle of Laennec, the grey hepatization of Andral, there are often here and there cavities containing a curdy pus, and communicating with the ulcerated bronchi. There are also occasionally found in it circumscribed indurations and tubercles of older date; but in other instances no other form of chronic lesion is present, and the lung has the appearance of inflammatory engorgement in some parts, of common red hepatization in others, whilst other portions of the same consistence have the opaque yellowish colour of tubercle; and these conditions pass by such imperceptible gradations into each other, that it is impossible to avoid the conclusion that they are parts of the same process. And can we wonder that vessels of the inflamed parenchyma of the lung should at the same time pour out in some parts organizable lymph, in others tuberculous matter, which is the same as albumen, only not organizable—can we wonder, I say, when we see a similar variety produced upon the inflamed pleura, where some portions are covered with good lymph, others with a curdy matter like tubercle, whilst many albuminous particles, also in an unorganizable state, are thrown off with the liquid into the sac? So also in the very masses of coagulable lymph that an inflamed pleura or peritoneum throws out, there have been sometimes found distinct purulent and tuberculous deposits: and why should we wonder at this either? Lymph, pus, and tubercle, are the same albuminous matter, and differ from each other in mechanical condition and susceptibility of organization, rather than in their chemical nature. According to the microscopic researches of Gendrin, part of which I have myself been able to confirm, lymph is composed of regular globules, which by a vital attraction string themselves into fibres and films and may become organized and form membranes. Pus consists of larger and less regular globules suspended in serum; but these globules have no vital attraction for each other, and remain loose and consequently insusceptible of further organization. Tuberculous matter is wholly devoid of organic form, its particles even not being globular, but irregular, like those of mere dirt or clay, and it must remain where formed, a dead inert mass, until decomposed by chemical agency, or changed by the operation of the surrounding tissues. You can readily perceive that these different properties, although possessed by matter chemically the same, and from the same source, must lead to all that variety of results which we know to follow organizable, purulent, and tuberculous deposits. But you are not to suppose that the characters of these matters are always distinct; that lymph is always equally

organizable, or perfectly free from the greenish colour and disintegrating globularity of pus, or even from the lifeless curdy particles of tubercles; nor that tuberculous matter shall not often contain flakes or films of imperfect lymph. The diffused tuberculation or infiltration of the lung from inflammation, that we have just been speaking of, generally presents a matter in this transition state. It is neither good organisable lymph, nor is it wholly unorganised tubercle; and the albuminous effusions on serous and mucous surfaces not unfrequently present such an intermediate state, that it is difficult to determine to which class they most belong. I have again strongly to repeat what I long since told you with regard to the inflammatory secretions of mucous and serous membranes—*that lymph, pus, and tubercle, pass by imperceptible gradations into each other.* The history of the intermediate products has yet to be more fully studied; and it is a subject of immense importance, for they probably constitute those forms of phthisical lesions which it is most within the power of medicine to control.

But lastly, we have found that tuberculous matter is sometimes deposited in tissues and on surfaces bearing no marks of inflammation or other disease. The structures thus affected are commonly those either very vascular naturally, or peculiarly subject to congestion of blood, such as the bronchial glands, the lungs of children, the spleen of monkeys, &c.; and viewing tuberculous matter as a deposit of unhealthy fibrin from the blood, we see a reason, as Dr. Carswell observes, why it is most likely to be found in those organs where the blood accumulates or passes slowly. Whatever may be the cause which determines the deposition of tuberculous matter in these cases, we know that pus also is sometimes secreted in parts unaffected with inflammation, as in the purulent deposits in the viscera after great surgical operations; and in the profuse discharges of matter from the bronchial membrane, which is found after death to be paler than usual: nay, pus has been occasionally found in the centre of fibrinous coagula in the heart, when no purulent matter could be discovered elsewhere in the body. So, likewise, tuberculous matter has been met with in the blood itself, within coagula in the spleen and in the heart, and in fibrinous concretions within the blood vessels. This circumstance tends to show that the fibrinous portions of the blood are liable to be converted into tubercle, independently of any action of the vessels; they lose their vitality, and may in proportion be merely deposited in tissues, or on surfaces without the presence of any irritation. We are thus again, as in case of the grey indurations, led to trace to the condition of the blood the cause of consumptive disease of the lung; and it is probably a diseased state of this fluid that constitutes what is called the tuberculous or scrofulous diathesis, in which there is a tendency by vessels in different degrees of activity to deposit tubercle instead of lymph: and when this diseased state exists to a great extent, the tuberculous matter is excreted from the blood without any increased vascular action, but merely as an accompaniment of the natural secretion of a membrane, or instead of the

ordinary nutrient deposit of a tissue ; and thus it may be either an external or an interstitial deposit. Whatever in such cases determines the first deposition of tubercle in a tissue, will with greater facility affect its growth by the addition of similar matter to a ready formed nucleus. The tendency to the deposit of yellow tubercle, independently of irritation, implies a condition of the blood even more depraved than that which leads, under the same circumstance, to the formation of the chronic indurations ; it is an ulterior degradation of the fibrinous nutriment of the tissues, replacing them by a matter not merely inapt in texture and of lower vitality, but wholly destitute of life, and of the principle of organization. When, therefore, tuberculous matter is found in an uninfamed tissue, it may be looked on as a sign of a most decided constitutional taint. In such conditions of the system, tuberculous depositions may take place with great rapidity ; and as they are already almost ripe for elimination, the ulceration and destruction of the lung will soon follow ; but nothing can give development to tuberculous disease with such fearful speed, as the occurrence of acute inflammation in the pulmonary tissue. It is from this process in a highly tuberculous constitution, I believe, that the general tuberculous consolidation, called infiltrated tubercle, takes place. The matter deposited is often rather a mixture, or intermediate state, of lymph and tubercle, one product predominating in one part, the other in another ; but it is altogether beyond the reach of the sorbefacient remedies which avail in pneumonia to promote the absorption of simple lymph ; and if it do not destroy life by its solid interference with the function of the lung, it speedily runs in many points into softening and suppuration, and the patient is carried off by a *galloping* consumption. In this case the lungs are found more or less solidified, and on incision incipient cavities are seen almost in every part ; but there is no induration ; the most solid parts have scarcely more substance than a hepatized lung, and they even more readily break down under the fingers.

The development of the indurations is a work of more time, and their structure makes the destructive process which they induce more tardy ; nay, the diffused indurations that form the walls of softened tubercles and vomices seem to be a provision of nature towards the limitation of the consuming disease ; but under certain circumstances even these are formed to such an extent, and so soon, that the patient is destroyed by their first development. In other cases the first formation of indurations is not sufficient to prove fatal, but as some of these are becoming converted into tubercles and cavities, another eruption or crop of them (as Laennec calls it) takes place, and causes suffocation. I do not conceive, however, that in either of these cases the miliary granulations alone are sufficient to destroy life ; but there is commonly with them an effusion of serum or of mucus, which completes the fatal obstruction. We have not time to inquire into all the circumstances which may lead to the development of these indurations ; but I will mention to you two cases which I have lately seen, and they are not the only ones of the kind, which illustrate

well some of the more important constitutional and local causes of these formidable lesions.

A young woman was attacked with continued fever, which, after several weeks of considerable danger, left her in a state of extreme weakness, so that she could only lie on her back, in which posture she remained altogether for at least six weeks. As the fever left her she began for the first time to cough, her breath became shorter, her pulse did not lose its frequency, and her faculties remained in an obtuse lethargic state. She died about a month after the first commencement of the cough. On dissection, the whole anterior parts of both lungs were quite healthy, to the depth of from one and a half to two inches. The whole of the posterior portions were highly congested with fluid blood, those quite behind scarcely containing any air. But the remarkable point was, that *every part of these congested portions was thickly studded with miliary granulations, whilst not one was to be found in the anterior parts.*

A lad of about 16 years of age became the subject of an attack of pleurisy of the right side, having previously enjoyed good health. It was inefficiently treated, and had become chronic when I first saw him. He considered himself, however, cured of the pleurisy, and complained only of weakness, although there was dulness on percussion, no respiration, and slight enlargement of the inferior two-thirds of the right chest. The sound and respiration above were clear but bronchial, and that on the left side was puerile and without ronchus. An attack of bronchitis subsequently came on, and from that time till the fatal termination, a month after, there were mucus and subcrepitant ronchi on both sides, and the expectoration at length became muco-purulent. In the lower half of the right side there were between two and three pints of purulent serum containing many flocculi. The lower lobes of the lung were quite compressed, and adhered to the mediastinum, and by a pillar to the diaphragm. The upper lobe was not compressed, and a dense fibro-cartilaginous membrane bound it to the chest, and limited the liquid effusion. The left lung was throughout studded with miliary tubercles, and there were a few in the upper part of the right lung, but *none in its lower compressed portions.*

In both these cases great constitutional debility preceded the development of the indurations; in both they were formed only in those parts of the lungs, to which the blood had the freest access, and were absent in those which, under the influence of gravitation acting on a weak circulation in one case, and of pressure in the other, received an unusually scanty supply of blood.

It will be hardly necessary, after what you have heard, to discuss the question of the seat of the hard grey and the yellow tuberculous deposition. If tubercle be, as we suppose, a degraded condition of the fibrine or nutrient principle of the blood, we may expect it to be deposited wherever the nutritive or the secreting process is carried on—wherever lymph or pus is occasionally found—wherever, in short, blood-vessels run. Instead of being, as supposed by Dr. Cars-

well, most commonly deposited on free mucous surfaces, its appearance on them is less usual, and bears the small proportion of the liability of these surfaces to the plastic inflammations. Lymph is sometimes, but rarely, secreted by mucous membranes; and it is about as common to find tuberculous matter, or a degenerated fibrin akin to it, in the air-tubes or cells, when the coats of these are entire. It is more common on the serous membranes; but it is much more usually (but not, as Lombard maintains, exclusively) deposited in the interstitial cellular texture of organs, especially those which are highly vascular, and in which blood is apt to stagnate or accumulate. I cannot at all assent to that most mechanical notion of Dr. Carswell, that tuberculous matter is, in the early stages of the disease, secreted in equal abundance from all parts of the mucous membrane, and that the only reason why it accumulates sooner and more in the upper lobes, is that their smaller extent of motion prevents its perfect elimination from those parts. Were this true, how easy would the diagnosis of consumption in its earliest stage be! for every atom of tubercle retained in the upper lobes there would be masses expectorated from the lower; yet we find that it is very rare to see any expectoration in the earliest stage, except that of a thin transparent phlegm. More probable is the opinion of Broussais, that the upper lobes are the first and most extensive seat of tuberculous change, because the bronchial tubes there are shorter, and inflammation more readily passes along them to the cells. But I apprehend the real reason of their peculiar liability is in the greater abundance of interstitial tissue in them than in the lower lobes. The bronchi, instead of being lengthened out into mere membranous tubes before they terminate in cells, divide immediately and abruptly into short branches and cells, and the delicate vesicular structure is thus mixed up with a good deal of the interstitial cellular tissue which every where surrounds the earlier bronchial divisions. The smaller capacity of motion possessed by the upper lobes of the lungs may, too, have a share in disposing them to become the seat of tubercular deposit, not by permitting it to accumulate, but by causing bronchial obstructions to the respiration, favouring those congestions of blood, &c., which we have seen, are circumstances peculiarly favourable to the formation of tubercles. It is not the yellow tubercle only that predominates particularly in the upper parts of the lung; I should say that it is rather the grey indurations, which become afterwards converted into yellow tubercle. Primary tuberculous deposits are nearly as common in other parts of the lung as at the apex. But if we regard the induration as an organized tissue, formed of albuminous matter of low vitality, but still above that of tubercle, we render its production referrible to the same causes as those which engender tubercle, and sufficiently explain the affinity of one lesion to the other.

I have described the ordinary changes of tubercle, from its primary deposition to its softening and evacuation, and the formation of an ulcerous cavern. These caverns become, if life lasts, lined with a deposit of a mixed nature, but with an albuminous lymph for its

basis ; and this is commonly mixed with tuberculous and purulent matter. Hence it seldom adheres long, but is broken up, detached, and expectorated. When the constitutional powers are stronger, and the lungs less extensively diseased, the coating of the cavity is susceptible of organization, and in time forms a fibrous, or fibro-cartilaginous membrane, which pretty smoothly lines the cavity. If the cavity communicate freely with the bronchi, it will be kept by the pressure of the air from any considerable contraction, to which it naturally tends ; and in some instances we have reason to believe that the tubes do become obstructed, and that the contraction of the membrane ensues, and tends to obliterate the cavity. Such contracted cavities are now and then met with, but scarcely ever quite empty ; they contain more or less of a pale-coloured plaster-like matter, which consists chiefly of carbonate and phosphate of lime, and sometimes contains earthy concretions. The contraction is evident from the puckering of the pulmonary tissue visible on the pleural surface near the cavity, and the adjoining vesicles are generally dilated to fill up the space. The cretaceous matter is probably secreted by the fibrous false membrane ; but it may have been originally more of the character of tubercle or pus, and being unable to escape, the animal part has been absorbed away, and the earthy insoluble salts are left behind, and accumulate from successive depositions. I have seen a similar matter in the remains of old pleuritic effusions ; and perhaps we may associate with it the osseous plates that are deposited in adventitious fibro-cartilaginous membranes, on the pleura, pericardium, and great vessels. This earthy deposit is, however, sometimes connected with an earlier stage of the tubercular formation. I have more than once, in lungs not extensively diseased, met with pale yellowish tubercles, composed of concentric layers of almost cartilaginous hardness : in another part of the same lung these layers are broken or loosened by a plaster-like gritty matter of a calcareous nature ; and in another part a whole tubercle may consist of this matter, sometimes containing concretions, and having only a few flakes of albuminous matter in it. This is still more distinctly a specimen of what is commonly called the atheromatous structure, which especially invades the coats of the arteries, and the fibrous parts of the valves of the heart, where it often proceeds to ossification ; and I think that it is to be classed with the grey and yellow tubercle, in so far as it is another variety of matter very low in the scale of organization, and formed of lymph of defective vitality. In these latter cases, there is no puckering or trace of contraction about the tubercle until it has evacuated its contents, which it is very slow to do, since it has not the tendency to soften and cause ulceration that makes common scrofulous tubercle so destructive. Hence I have several times seen a few of these tubercles in lungs otherwise healthy, the individuals having died of some other disease.

Our time does not permit us to enter into many other particulars respecting the pathology of tuberculous disease, which would further illustrate the views which we have taken, and enlarge your know-

ledge of the facts connected with the subject. I refer you especially to the papers of Professor Alison, in the first volume of the Transactions of the Medico-Chirurgical Society of Edinburgh ; to Andral's Pathological Anatomy ; Louis' Treatise on Phthisis ; and Lombard's Essay on Tubercles.* You will also find much valuable observation in the articles *Tubercle* and *Tubercular Phthisis*, in the Cyclopædia, by Drs. Carswell and Clark ; but I doubt that you will remain satisfied with the view which the former takes of the pathology of pulmonary tubercles, however ingenious it may appear to be at first sight. In referring you to these different writings, where you will see other opinions stated, and other explanations given, I would ask you to bear in mind the general view that I have given you of the nature of tuberculous deposits, whether grey, dark coloured, or yellow ; that they are formed of lymph of defective vitality, and are either very low in the scale of organization, or altogether beneath it ; that whether arising from secretion or textural nutrition locally disturbed by chronic inflammation, or from these processes having to act, in their natural state, or excited by vascular action, on a blood defective in vitalized fibrin, it is this want of vitality in the deposits which determines their character and course, and leads to the formidable lesions which are induced by them.

LECTURE XXII.

Diseases of the Parenchyma of the Lung (continued).—Phthisis Pulmonalis.—Pathological History (continued).—Recapitulation to explain the Changes.—Origin of Phthisical Disease ; from Inflammation ; from Constitutional Disease ; Symptoms of Phthisis.—First Stage, Irritation and Obstruction ; Second Stage, Tuberculous Deposition ; Third Stage, Softening, Ulceration, &c.—Complications.—Physical Signs of Phthisis.—First Stage, Percussion and Inspection.—Stethoscopic Signs.—Difficulty and Importance of the Diagnosis.

WE had, in the last lecture, so many points to discuss, and so many views to examine, that I think it right to aid your memory by once more stating a summary of the pathological changes which are produced in the lungs by phthisical disease, with a reference to their analogies to other lesions.

1. Induration, whether granular or diffused, caused by the deposition in the tissue, of animal matter of low organization, under the influence of chronic inflammation, or of defective vitality in that part of the blood which maintains the nutrition of the tissues.

*To these may be now added a very important paper on the morbid anatomy of 100 cases of phthisis, in the number of the Edinburgh Medical and Surgical Journal for last January, by the late Dr. Home, jun., whose premature death has deprived the profession of a member of whose talents for observation this first contribution gave the highest promise.

2. The conversion of this induration into crude yellow tubercle, by the substitution of a friable albumen wholly destitute of vitality. This change has its parallel in the increase of fragility and opacity, which precedes the conversion of effused lymph into pus. Sometimes the indurated tissue is destroyed by direct ulceration, without the formation of yellow tubercle.

3. The formation of vomicæ by the softening of crude tubercle, and the original deposition of soft tuberculous matter. These bear analogy to suppuration, and the formation of purulent depôts.

4. The evacuation of vomicæ, and the formation of ulcerous cavities, which is like that of fistulous ulcers from abscesses.

5. The hardening of tubercle without discharge, into cretaceous tubercles; like the conversion of abscesses into indolent tumors.

6. Accidents contingent on the preceding changes, such as hæmoptysis, from the obstruction or ulceration of blood-vessels. Perforation of the pleura, and consequent pleuritic pneumothorax. Inter-current inflammations, bronchitis, pneumonia, ulceration and other changes in the bronchi, trachea, and larynx.

Besides these, various changes are commonly produced in other parts of the system, such as tuberculous disease in the mesenteric, and other lymphatic glands, spleen, liver, brain, ovaries, and other parts, and tubercle, combined with induration, or some low form of organized structure on the serous membranes, the pleura, peritoneum, membranes of the brain, &c.; inflammation and ulceration, probably proceeding from tuberculous deposits in various parts of the intestinal mucous membranes; chronic inflammation of the joints, and brown softening of their synovial membranes; diseased nutrition of the interosseous ligaments; greasy degeneration of the liver; and general wasting of all the tissues.

Now, instead of giving you a detailed account of the symptoms of phthisis, for which we have not time, I shall endeavour to class them according to the pathological stages of the lesions that give rise to them. But before any of these lesions are actually formed, we have found it necessary to assume that there is a cause, either local or constitutional, or partaking of the character of both, which precedes them. So, also, if we attend to different cases, of pulmonary consumption, we shall find some in which the individuals had enjoyed very good health until they were attacked with one or more severe colds, or inflammations of the chest, or a fever accompanied by pectoral symptoms sooner or later, after which the phthisical disease commenced. In others, again, the cough and other symptoms begin very gradually, without any very obvious cause, and with as little apparent external reason soon increase to a serious extent, and the consumption runs a rapid career. In a third class of cases, the patients have been out of health, in a debilitated or cachectic state, before the commencement of the cough and other local symptoms, which become suddenly developed after exposure to cold, the stoppage of an habitual evacuation, or some other cause likely to occasion local irritation or plethóra.

In the first class of cases we have the development of phthisis from local inflammation, without any evidence of prior constitutional disorder, unless an hereditary predisposition, which may be traced in some of these cases, may be considered as such. The acute inflammation, whether pulmonic, pleuritic, or perhaps bronchial, imperfectly treated and only partially subsided, passes into a chronic form, and either immediately develops phthisical indurations in the lungs, or by lowering the vital powers generally, leads to their formation from perverted nutrition, or from the irritation of any fresh exciting cause. Under any of these circumstances, chronic inflammation, either by its own local effects, or by its depressing influence on the constitution, or by both combined, becomes a sufficient cause of pulmonary consumption : and, as you may suppose, consumptive disease originating in this way, is sometimes more limited in its extent, and manageable in its course, than that first arising from a constitutional disorder. As the cause is more local, so the lesion is more confined to a part ; and we see instances of it in the very partial indurations or tuberculous depositions which we frequently meet with in the lungs of elderly persons who die of other complaints. In at least half the instances in which I have thoroughly examined the lungs of persons in London and in Paris, above the age of forty, without reference to the disease which caused their death, I have found some traces of those lesions which, occurring in greater extent, constitute phthisical disease. You can very easily discover these by passing the lungs quickly between your fingers, and when you feel a hard body, on cutting into it, it will be found to be either a grey induration or a yellow tubercle, or the remains of one ; whilst nothing in the rest of the body betokens any scrofulous taint. Yet the same local causes that produced these solitary tubercles, may engender many ; and the greater the number that the local cause thus develops, the more effect will it have on the constitution, which becomes in a manner inoculated by it. Hence, therefore, also, out of a local cause, such as a latent or neglected pneumonia, pleurisy, or peritonitis, may arise a general tuberculous disease, involving, more or less, the whole system. Among the local causes of consumption are to be reckoned, also, the habitual inhalation of fine solid particles, which is contingent on certain occupations, such as those of needle-pointers, dry grinders, stone-masons, miners, colliers, and the like. The dependence of the lesions, in these cases, on the mechanical irritation of the inhaled particles, is sufficiently proved by the presence of these particles in the indurated lung, which in the case of colliers is completely blackened by them. With these cases may be associated those of phthisis, produced in the experiments of Saunders, Cruveilhier, and others, in the lungs of animals, by the injection of mercury into the air-tubes, or into the blood-vessels.

In the second and third class of cases, which are unfortunately the most numerous, the disease is certainly constitutional ; and the cachectic condition of the system, as well as the circumstances that seem to occasion it, fully correspond with the pathological views

which we have taken of the origin of tubercle. Imperfect nutrition, whether from deficient or improper food, or from a permanently disordered state of the digestive or assimilative organs; unhealthy air, whether from closeness, humidity, or impurities; long continued exposure to cold (as from insufficient clothing), not intense enough to produce inflammation; depressing passions, such as disappointed love, anxiety or distress from reverses of fortune, or other severe calamity; venereal excesses; profuse and very weakening discharges; adynamic fevers; irregularities of the uterine function, especially those that lead to chlorosis; the sudden suppression of habitual discharges, or of long-established cutaneous eruptions,—these, singly or combined, are the most common causes of the constitutional origin of tuberculous matter; they all tend to destroy the balance of the functions, and diminish the tone of the system, and with it that rich fibrinous and vital condition of the blood, by which proper nutrition and the organic functions are sustained. The impoverished blood, defective in that vital albumen with which the tissues are fed and renewed, deposits in its stead a degraded matter, imperfectly or not at all organizable, like that resulting from the lower degrees of local irritation or inflammation. The lungs, the lymphatic glands, and a few other organs, become the first seat of these deposits, because their textures are in freer relation with the blood than those of other parts are; and if there be in these organs also a congestion, an irritation, or an inflammation, the deposition becomes more extensive and rapid in proportion, although, if the inflammation be acute, it may also produce organizable lymph, together with the degraded albumen, tubercle. And mark how the natural activity of the nutritive process will determine the rapidity of the tuberculous deposition, and the progress of the disease. In young persons nutrition is most active; so is consumption spontaneous and rapid; and at no period is this more manifest than between the ages of eighteen and twenty-five, when growth becomes completed, yet the vessels and their blood do not immediately lose their habit of deposition. But throughout the whole period of childhood and youth tuberculous disease is very common, and is more easily engendered than when nutrition is less active, under the influence of the causes which I have enumerated.—You can understand, too, why this morbid nutritive activity, this disposition to deposit albuminous matter (in such cases inorganic, tuberculous), should be shown in women after the completion of utero-gestation, and in persons on the speedy healing of large suppurating wounds—circumstances which, as long as they continue, are known often to suspend the progress of consumptive disease. For a further account of the constitutional causes of tuberculous disease, and for the only good description of the condition of the system which precedes its development, I must refer you to the work of my friend, Dr. (now Sir) James Clark, on Pulmonary Consumption and Scrofula, by far the most complete work which we possess on the subject.

Let us now quickly enumerate the symptoms of pulmonary con-

sumption in the order of the stages of the lesions which produce them.

First stage.—The indurations are accompanied as well as preceded by various irritations, both local and general. Of the local irritations one of the earliest is *cough*, which at first is generally slight, and merely hacking, but more or less constant; either dry, or attended merely with a thin transparent mucous expectoration. Another occasional sign of local irritation is *pain* in the chest, which is commonly referred to the sternum, and is rather a soreness, or an unusual sensibility to cold or exertion, than a continual pain. Of the more general irritations, *quickness of the pulse* is the most constant, but it is not universal. The quickness is frequently not uniform at first, but depends on trifling causes of excitement, and the pulse may be very slow and weak at intervals; but as the organic lesion increases, it gradually becomes more constant, and is accompanied by an irritated state of the other functions—a general febrile state. But even then there is not power enough in the circulation to maintain a general or constantly increased heat; it is manifested mostly towards night, after the accumulated excitements of the day; and it is generally most felt by flushing of the face, and heat in the palms of the hands and soles of the feet, where the thickness and hardness of the cuticle prevent the perspiration and evaporation which moderate the temperature of other parts. Like other weak febrile movements, this terminates by perspiration more or less profuse, which, occurring in the night, leaves the pulse lowered, but the frame weakened and exhausted in the morning. It is only in the severer cases that this general irritation, or *hectic fever* as it is termed, becomes marked at this early stage of the disease. There is often also gastric irritation, with the tongue white and red at the edges, thirst, costive bowels, and turbid urine. These symptoms are perhaps more remarkable in this than in the after stages, when the irritation is more confined to the organs of respiration and circulation.

The symptoms from *obstruction* comprehend those from the *obstruction to the air, to the blood, and to the motion of the lungs*. The indurations, granular and diffused, when extensive, *by obstructing the passage of air* to the lungs, occasion the short breath felt at first only on exertion, so common even in the early stage of consumption. Nay, cases happen in which an abundant formation of miliary tubercles, together with the œdema or bronchorrhœa that they excite, prove fatal in their first stage, by the obstruction which they cause to the passage of air. We have seen that tubercles, or any other partial obstructions, may become a cause of pulmonary emphysema, or dilatation of the air-cells, in the manner which I have already explained to you. The indurations, by *obstructing the blood-vessels*, give rise to many formidable pathological effects. They may thus cause sanguineous congestion, hemorrhage, inflammation, œdema, gangrene, and atrophy of the pulmonary texture, hæmoptysis, profuse bronchial secretions, effusion into the pleura, disease of the heart, and so forth; and the symptoms which these lesions pro-

duce may be variously grouped in the history of different cases of phthisis. The hæmoptysis occurring in the early stages of consumption is generally from this cause ; and it is a serious symptom, not only because it may endanger life by the loss of blood, or direct suffocation, but also because it is often accompanied by hemorrhagic consolidation and ruptures of the texture of the lungs, which tend to accelerate the disorganizing process, and promote the further deposition of tubercle. In some instances, however, hæmoptysis is followed by decided relief to the dyspnœa and cough, having removed a congested state of the vessels. The *obstruction to the motion of the lungs* may be caused by the same circumstances that impede the free admission of air to them, but in case of extensive solid deposition may also result from their mechanical resistance to the respiratory movements ; and this not only constitutes a physical sign which we shall consider afterwards, but it also keeps the intervening unaffected texture in a fixed state, liable to consequent congestion and further deposit, and adding further to the incapacity of the organs.—When once the integrity of a nicely adjusted apparatus like that of respiration is extensively injured, you see how the mischief becomes extended ; disorder begets disorder ; and unless the counteracting or reparatory powers come soon into action—unless the indurations are diminished, or the vessels blocked up—the whole of that part of the lung may soon become a solid mass. Thus, I believe, sometimes arise those extensive masses of induration that are so commonly met with in the superior parts of the lung.

Second stage.—On the conversion of the grey or dark red indurations into crude yellow tubercle, and the original deposition of this matter, in addition to the symptoms of irritation and obstruction, which still continue, there are indications of an increasing cachexia, a depression of the functions. The pulse loses strength ; the fever is of shorter duration ; and the sweats are more profuse ; except at times of excitement, the colour of the cheek fades, or is reduced to the circumscribed hectic patch ; the expectoration becomes more abundant, or less thin and transparent, and particles of curdy or cheesy matter can sometimes be detected in it. But all these symptoms more properly and fully belong to the

Third stage, in which the tubercles become soft, partially or entirely liquid, and are evacuated by the aid of the secretion and ulceration of the adjoining textures. Then comes on, in addition to the other symptoms, the copious and heterogeneous expectoration of pus, mucus, softened and occasionally solid tubercle, blood, shreds of lymph, rarely portions of pulmonary tissue, sometimes very fœtid. Then follow the usually constitutional concomitants of extensive unhealthy suppurating ulcers, confirmed hectic and marasmus, rendered here more pronounced by the importance of the organ affected, and the relation which it bears to the process of sanguification. Then are the dyspnœa and cough increased by the continual discharge of matter into the air-passages, and by the extension of the diseased depositions and ulcerations of the tissue. Yet it is a curious circumstance,

that these symptoms are often inconsiderable in proportion to the terrible extent of the organic mischief that has been produced: the dyspnœa is not painful, like that of asthma; it is a state of breathlessness, rather than of distressing oppression; the cough is not so violent as in chronic bronchitis; the pain may be but slight or none at all; the countenance, though thinned, hectic, and with the sharpened nostrils habitually moving at every breath, may have a clearness in it, with a colour in the lips and a brightness in the eye, which are never seen in other diseases; and the frame of mind is often in the same unconscious and hopeful state, indicating a degree of freedom from those painful struggles in which the vital powers commonly contend with other serious disorders. Now I apprehend the chief reason of this exemption from suffering lies in a sort of balance that is produced among the injured functions. The available part of the lungs is reduced to a surprising extent; but so is the mass of blood that has to pass through it. The free expectoration and the colliquative discharges are continually bringing down the bulk of the circulating fluids to the lessening capacity of the remaining lung. The night-sweats especially are a periodic discharge of the amount of fluid which is beyond what the reduced system of blood vessels can quietly hold; they generally cease when the fluid ingesta are carefully reduced. So the secondary pulmonary irritations, congestions, and inflammations, are continually relieved by the purulent expectoration; it is a safety-valve which gives vent to these before they can cause distress; and although the destructive process is here perpetually proceeding, the lungs are decaying, the body consuming, and the strength failing, yet it is all by even degrees, a *facilis descensus*; the thread of life dwindles away, fibre by fibre, without struggle or shock; and gentle is the parting of the last filament, when the body drops to earth and the soul rises to eternity.

But the progress of consumptive disease is not always thus painless and unharassed: the sufferings from dyspnœa, cough, pain, heat, and feelings of extreme weakness and sinking, are sometimes very severe. There are those whose animal sensibility is more acute than their organic life is active; to such persons any disorder is distressing; and, even in consumption, the cough, the pains in the chest and sides, the alternate chills and heats, the oppression of dyspnœa, the faintness of debility, besides innumerable pains and aches of various parts of the body, are perpetual sources of complaint. But without an unusual degree of sensibility in the subject, the course of consumptive disease may be rendered rough and painful by what may be called the accidents or irregularities contingent upon it. Intercurrent congestions, hemorrhages, and inflammations taking place in the lungs or their investing membranes, may give rise to symptoms of these several acute lesions, superadded to those of phthisis. I have said that a free expectoration tends to prevent these accidental complications: and, accordingly, their occurrence is often preceded or accompanied by a suppression of this discharge or an alteration

in its quality.* But there is an accident which especially tends to ruffle and hasten the course even of the quietest forms of consumptive disease; this is perforation of the pleura, and the consequent pneumothorax and acute pleurisy which it produces. As I have already described this lesion, and the symptoms which it induces, I need not now dwell on it; but I would remind you how characteristic the sudden increase of dyspnœa and accession of sharp pleuritic pain must be in the cases that were before the most insidious, and how much the addition of these lesions must increase the distress of the patient, and hurry him towards his grave. I think that perforation of the pleura occurs generally in cases where the constitution is decidedly tuberculous. It implies a want of that self-preserving energy by which the mischief of ulceration is limited by the timely effusion of plastic lymph. This is a part of the nutrient function, and I suppose this is commonly more active in women than in men; for if I can judge by my own experience, perforation of the lung very rarely occurs in females: I have never met with one instance, yet I have seen at least twenty cases in the male sex; and, as far as I can recollect, the cases recorded by authors are chiefly of that sex.

Other symptoms unconnected with the chest occasionally attend pulmonary consumption. The larynx is very often diseased in phthisis; the hoarseness or loss of voice being an early symptom, and taking attention from the real and chief seat of disease. The lining membranes of the larynx, and the vocal ligaments, are in such cases thickened, eroded, and ulcerated, and the cartilages and bones may ultimately become carious or necrosed. There is even in these lesions something of the intractable character of tuberculous ulceration; therefore beware of *laryngeal phthisis*, for if it be not already complicated with tubercles in the lungs, they may be formed at any time, for the leaven is in the system. The disorder of the digestive organs, which, in the earliest (the irritative) stage, had something of the gastritic character, with red-tipped tongue, thirst and indigestion, perhaps some tenderness in the epigastrium, commonly passes off when the pulmonary irritation is relieved by the discharge. At this period, however, the bowels often become disordered, and constipation and diarrhœa alternately prevail, dependent on inflammation and ulceration, often complicated with tuberculous depositions in the follicular structure of the mucous membrane of the intestines. The mesenteric glands, too, become frequently involved in similar disease; and thus arise additional causes of exhaustion and atrophy in the colliquative discharges, and the obstruction to nutrition that ensue. The character of the alvine secretions sometimes shows a deficiency of bile; but this is a symptom which more frequently precedes phthisis than accompanies it. Sometimes there are great tenderness and even pain in the abdomen during the whole course of the disease, with occasional exacerbations; these symptoms generally depend on

* Dr. Stokes has made similar observations with respect to the influence of expectoration, in relieving the local symptoms of phthisis.

granular or tuberculous depositions on the peritoneum, combined occasionally with inflammation of the membrane. More rarely, tubercles occur in the brain or spinal marrow, or their membranes, and cause symptoms of mental disorder, convulsions or paralysis. Acute hydrocephalus seems to have some further connexion with scrofulous or tubercular disease; for it sometimes co-exists with tuberculous depositions in other parts, although there be none in the brain or its membranes. I cannot now dwell on the details of other symptoms arising from the irritations or obstructions, the weakness and the wasting, which tubercles bring in their train. The emaciation in the last stages is great, but that from scirrhus of the stomach, or *tabes mesenterica*, is greater. There is a blanching with the emaciation, that is more remarkable than its degree; the blood vessels are reduced, as well as other textures, and you scarcely ever, in tuberculous consumption, see the redness of the knuckles and distinctness of the veins which accompany even greater degrees of emaciation from chronic diseases of the abdomen.

Let us now pass on to the consideration of the physical signs of pulmonary consumption, tracing them in the different stages of the textural lesions which we have described. In proportion as these lesions are of great or small amount, or are concentrated within a small space, or are scattered widely through the lung, they will produce more or less appreciable signs. Thus the miliary indurations, even in considerable number, may be scattered through the tissue of the lung without producing any distinct diminution or change in the respiratory sound, or in the resonance of the chest on percussion. Sometimes there is a general sub-mucous or sub-crepitant rhonchus; but this proceeds less from the tubercles than from the secretion which their irritation causes in the adjoining bronchial tubes: it is the sign of a partial bronchitis, therefore, and can only be taken in evidence of the probable presence of tubercles, when it continues permanently day after day, instead of tending to become sibilant, and to pass off as the rhonchi of common bronchitis do. But it seldom happens that even the early indurations are equally scattered through both lungs. Their tendency is to accumulate in greater numbers or in little clusters near the apices of the lungs, and generally more on one side than on the other. Here, then, will be a concentration of effect, and a difference between the two sides of the chest; and if we explore the corresponding region on the exterior, which are the clavicles and the space immediately beneath them, and the upper ridge of the scapulæ, we may find differences in the sound on percussion, or those of respiration, and the voice, which, according to known principles, may be interpreted as signs. The clavicle on one side, when struck lightly downwards on its middle, yields a sound duller than that on the other; and especially if this difference extends to the other parts just mentioned, it is exceedingly probable that there is tubercular consolidation of the lung in that part. Great care must be taken to strike both clavicles at the same point, or both infra-clavicular spaces in the same mode, or the comparison will not be a fair one. You

should, to avoid error, have the parts quite uncovered, and tap either directly, or on a finger, on corresponding points of both clavicles. Below these bones, gentle percussio with the flat fingers, mediate or immediate, generally succeeds best ; but various kinds of percussio should be tried in doubtful cases, and in different stages of the respiratory act—on a full breath, and after exhausting the lungs. You are to remember that the consolidations may be merely granular and scattered, and that they would not have body enough to affect the vibration of the walls, if the stroke of percussio be hard ; nay, sometimes the gentlest possible *patting* of the space under the clavicles is the only mode in which any difference can be detected. In the posterior region, however, and above, on the scapular ridge, considerable force is required to get any pectoral sound at all, and this must be used by mediate percussio on a finger laid along it ; comparing, as usual, the two sides. Always seek for differences in the sound where differences ought not to exist ; and vary your mode, place, and force of percussio, at different times ; but let these be carefully the same in each act of comparison. When the infra-clavian spaces fail to give any signs of disease, try the parts below, at the sides, and in the back. Between the scapulæ is not an unfrequent seat of dulness, especially in children, where the disease occupies the bronchial glands.

You should not omit to use your eyes, too, in scrutinizing the condition of the chest, by making the patient breathe fully, whilst you stand in front, in a good light, and watch and feel its motions. It requires more consolidation than is usual, in the doubtful stages of the disease, to produce any considerable irregularity in the shape or movements of the chest ; but we can often perceive a slight difference between the two sides: the upper ribs do not move quite so much on one side as on the other. The collapse of the infra-clavicular space, so remarkable at later periods, does not take place at this stage.

Thestethoscopic signs are more delicate, and perhaps more equivocal than those of percussio. The indurations may form slight partial obstructions to the passage of air, and thus cause a permanent slight wheezing, whistling or roughness in the respiratory sound, not removed by full inspiration or cough. If more numerous or extensive, they may transmit the sound of whiffing, bronchial breathing in parts where naturally the vesicular only is heard, whilst the soft vesicular breathing is impaired in its force. The sound of expiration may become unusually audible, and sometimes equals that of the weakened inspiration, which you know is almost the only sound in purely vesicular respiration. When, as it often happens, the partial indurations are accompanied by a dilated or emphysematous state of the neighbouring air cells, the sound on percussio will be less altered than usual ; but the breathing will be more whiffing, or more obscure, according as the dilated cells are more flaccid, or more rigid than usual. When more rigid, they diminish not only the sound of respiration, but also the motion. The sound of the voice is transmitted by the indurations in an unusual degree, being sometimes only

a diffused resonance, but sometimes quite a little voice under the clavicle ; but without the articulation of the oral voice. So also the sounds are occasionally transmitted with uncommon clearness from the subjacent arteries, being either double from the second sound of the heart, or single from the mere impulse ; and now and then the single pulse is accompanied by a whizzing or blowing, confined to that part indicative of partial obstruction of the subclavian artery, from the pressure of indurations at the apex of the lung.* I have known it to intermit regularly, occurring in two or three pulses during the first part of expiration.

Now all these stethoscopic signs derive their importance directly from the situation in which they are heard, and from their comparison with other parts. There are often bronchophony, you know, and bronchial respiration, naturally, near the sternum, between the scapulæ and in the axillæ ; therefore you must not place confidence in such signs in that neighborhood, unless they be much more distinct on one side than on the other ; or accompanied by dulness on percussion. You may trust to them more towards the humeral end of the clavicle ; the angle formed by this bone and that of the shoulder is the proper stethoscopic corner, and the signs heard there are the most unequivocal ; but even here a permanent discrepancy between the two sides gives the surest indications of disease ; for there are many modifications of the natural sounds heard there.

I must not disguise from you that this is a very difficult point of physical diagnosis, and requires much experience and nice perception on the part of the auscultator. But you are not on this account to be discouraged from attempting it ; on the contrary, it the more deserves and requires your study ; and if you will only devote proper attention to the method, and take every opportunity of practising it, you may in time acquire the means of detecting phthisical disease in its earliest and least intractable stage, and you may thus be enabled to save many valuable lives from otherwise inevitable destruction.— You must not neglect attention to the general symptoms ; for besides the assistance which they give in the diagnosis by the character of the cough and pulse, they are, as I have often told you, of the utmost importance in guiding the practice, which is to be adapted to the state of the whole constitution as indicated by them, as well as to the local disease, which the physical signs alone may have discovered.

The cases which we have to deal with are commonly those in which there is a cough, which, from its duration, its unusual character, or from its being a new symptom in the subject, excites some apprehension. Is this a common cold ? Is it a “nervous cough,” a “stomach cough,” or a “liver cough?” Or is there a permanent cause of irritation in the pulmonary tissue itself ? We first endeavour

* This subclavian arterial murmur has been noticed by Dr. Stokes as a sign of incipient phthisis. I have never met with it except in cases where percussion and the other signs indicated a considerable extent of consolidation.

to answer these questions by physical examination. If there be no tuberculous disease there will be none of the signs that I have just been describing, but those merely of bronchitis or catarrh; various rhonchi, sibilant, sonorous, and mucous, in various parts of the chest, generally in the middle regions, where the large bronchi pass; not so much in the upper parts, and never confined to them. Then you may inquire whether there are other symptoms which may justify you in considering the bronchial irritation as secondary, and referrible to an excitable nervous system, to a disordered stomach, or a congested liver; and if you find them, well and good; act accordingly. But my experience has led me to look with suspicion on all coughs which last an unusual time, until by repeated examination I have found no cause for them in the lungs. Many a consumptive case has been dressed up, even to its last stage, in the delusive names "liver" or "stomach coughs."

LECTURE XXIII.

Diseases of the Parenchyma of the Lung (concluded).—Pulmonary Consumption, Physical Signs (continued).—Signs of Softening and Evacuation of Tubercles.—Signs of Cavens, Pectoriloquy, &c.—Expectoration in the latter stages.—Signs of Intercurrent Diseases.—Process of Cure of Phthisical Lesions.—Treatment of Pulmonary Consumption.—First Stage; Antiphlogistic Remedies.—Sorbefacient Alteratives, Iodine, &c.—Constitutional Treatment.—Diet and Regimen.—Treatment of Symptoms.—Latter Stages; External Counter-Irritations, &c.—Constitutional Treatment.—Treatment of Symptoms.—Encephaloid Diseases in the Lungs.—Scirrhus in the Lungs.—Melanosis in the Lungs.—Spurious Melanosis.—Diseases of the Bronchial Glands.

THE conversion of the semi-transparent, grey or dark consolidation of the lung, into crude yellow tubercle, is a point only deduced from anatomical examinations. We have no signs of this change during life. I have before observed, that there is sometimes an abatement of the symptoms of irritation, which may perhaps be associated with this change; there may be at the same time an increase in the expectoration, and the submucous and mucous rhonchi become more marked. But the change to yellow tubercle can hardly take place without some augmentation of the consolidation; the indurations increase in extent, and some yellow tubercle may perhaps be deposited or secreted in other parts. Hence there is a fuller development of the signs of an increased density in the lung; the partial dulness on percussion becomes more marked; the respiration becomes more obscure or more bronchial, and it may be accompanied by a permanent fine crepitation. The vocal resonance may have also increased in degree and extent, and altogether the signs have become more localized, and therefore less equivocal.

But it is the softening and evacuation of tuberculous matter that

produce the most remarkable and cognizable changes in the physical signs ; and these too often give to the expectoration something of the precision of a physical sign. The sputa before may have been sometimes opaque and muco-purulent, as in bronchitis ; but they now become decidedly purulent, often sink in water, and if narrowly examined, may sometimes be found to contain particles of curdy or clotted matter, like cheese softened in water, which is tuberculous ; they are not foetid like the similar concretions from the tonsils. There may also be little streaks or even clots of blood ; but this is uncertain : there is generally, besides, more or less mucous, which gives tenacity to parts of the expectorated matter ; but on close examination it may be seen that some sputa are opaque purulent clots, almost without mucus ; and it is these that come directly from the cavities. Wherever these changes take place, generally under one of the clavicles, or above the spine of one of the scapulæ, there you may hear a clicking or bubbling sound, which is coarser, and gives you the idea of being produced in a larger space than any of the common sounds of these parts. And here you will see that this sign is more conclusive, the finer and more completely vesicular is the natural structure of the lung in the part in which it is best heard. In listening for this sound, you must seek for it in a long inspiration, or during a cough, which makes the air enter more fully and suddenly ; and at first you may hear no more than one or two *clicks*, from the entry of single bubbles ; but as the evacuation of the softened matter proceeds, and there is more room for the entrance of air, there is then a more continued gurgling or bubbling sound ; and this will be coarse and distinct in proportion to the extent of the vomica, and its communication with the air tubes. This gurgling or *cavernous ronchus* will also vary somewhat, according to the quantity and liquidity of the contents of the cavity ; becoming less crackling, and more whiffing as these diminish. When it is heard over an extended space, there are probably several cavities communicating with each other, and all containing more or less liquid. But I think it is not necessary to detail to you all the varieties that circumstances may produce ; when you know the principles of the phenomena, you can foresee and understand their varieties.

The softening and evacuation of the vomica being complete, there is left an ulcerous cavity or cavern, which becomes the seat of further phenomena. Even before all the liquid is evacuated we sometimes hear, in the corresponding part of the chest, with the gurgling a hollow whiffing or blowing sound ; and when the patient speaks, a sort of *snuffling* voice, interrupted, broken up by the gurgling. When the cavern is empty these pass into *cavernous respiration* and *pectoriloquy*. Cavernous respiration is something like what you hear on applying the stethoscope to the front of the neck, in the course of the windpipe ; but it is more circumscribed, and does not give you the same impression of a rush of air. You may get a better imitation of it by blowing into thimbles or shells of different sizes. It may present considerable variety, according to the size and shape of the cavity,

and the freedom with which the air passes into and out of it from the bronchi. When of very large extent, the sound becomes amphoric, like that produced by blowing into an empty phial, and precisely on the same principle. All these phenomena are better obtained with quick forcible respiration, or slight coughing, which increases the force and velocity of the passing air, and exaggerates all the sounds.

Pectoriloquy is another very striking sign of a cavity in the lungs. Its value was perhaps overrated by Laennec ; but I think it has been neither appreciated nor understood by subsequent writers. I formerly explained to you that the voice, although formed in the larynx, vibrates in full strength through the windpipe and its branches, until it becomes broken up and muffled in the smaller tubes and porous tissue of the lung. But if a cavity be formed in this parenchyma, communicating freely with the tubes in which the voice is strong, it will form a part of those tubes, and the vibrations will be continued in *system* from them to it ; and there may thus be heard near the surface of the lung a voice from the chest like that heard over the trachea ; its distinctness and intensity, however, being more or less perfect according as the cavity is adapted to receive the vocal resonance from the tubes, and to transmit it to the walls of the chest.—Laennec made an artificial distinction between the degrees of pectoriloquy, according to whether the voice does or does not give the impression of passing up the stethoscope with the stopper to the ear. In the *perfect* kind, the words are so distinct that it seems as if the patient had his mouth to the tube : where this impression is not produced, the pectoriloquy is *imperfect*. But this is only a difference of degree, the sound most perfectly transmitted *engrossing* the air in the tube with its vibrations ; whereas a less perfect transmission affects it more partially. I consider the *character of the sound* and its *circumscribed position*, a better distinction. The sound is not a mere vocal resonance, like the bronchophony from consolidation, which is often as loud or louder, and may pass up the tube ; but it is an articulate although indistinct speaking, and sometimes accompanies a loud whisper as well as vocal utterance. There is in it another feature which is characteristic, and distinguishes it from bronchophony ; it is accompanied or followed either by whiffs of cavernous respiration, which give the pectoriloquy a *snuffling* character, or by a hollow or fistular resonance, like that which you can produce on speaking at the mouth of the tube of a pan-pipe, the pipe of a large key, a shell, or any such hollow body. This accompaniment is sometimes observed when the pectoriloquy, or transmission of the articulate voice, is very imperfect ; but I have found it to be more distinctive of a cavity than the loudest vocal sound without it. I suppose that it depends on the same physical cause as the phenomena of the hollow bodies to which I have compared it ; the cavity in the lungs being in the same relation to the bronchial voice as they are to the oral voice. When the cavity is very large the accompaniment is more amphoric or bottle-like ; and if the communication with the

bronchi be at the same time narrow, the voice may scarcely be transmitted to it, but only excite in it a tinkling echo, a metallic tinkling, as in pneumo-thorax. All these hollow, fistular, or tinkling characters, may be also perceived in the breathing and cough, especially in the latter, but not in a proportionate degree; and I have known them to be scarcely perceptible but with the voice. These differences must depend on the relations of the cavity to the air-tubes communicating with it; if this open into them so as to catch the current of air passing in them, its interior will be thrown into vibration; otherwise the air within it may only receive the stronger and more pervading vibrations of the voice. So also, if there be much consolidation about and beyond the cavity, there may be very little passage of air in the tubes, and therefore but little cavernous breathing.

The circumscription of pectoriloquy is another of its peculiar characters, and by this it may generally be distinguished from the loud bronchophony of condensed lungs, which is diffused over some extent of surface. To observe these differences, it is quite necessary to limit the point of examination, by using the stopper in the stethoscope. By this means you can trace the precise boundaries of the pectoriloquy of a cavity; but when you try to find where the resonance of bronchophony ceases, you will fail to find any exact limits; it gradually loses force as the tubes become smaller, or the superjacent lung more porous. Pectoriloquy is most characteristic when it forms a *little island of voice* under a clavicle, and little or no sound is transmitted nearer the sternum. The pectoriloquous bronchophony of a lung consolidated by inflammation, or compressed against the walls of the chest by a liquid effusion, never has this isolated character, but is generally louder in proportion to the size of the tubes involved in the condensation. It is, however, true, that sometimes, in phthisis, the pectoriloquy is not thus circumscribed; for besides the cavities, there may be general consolidation of the lung, and consequently free transmission of the voice over an extent of surface.—Even in this case, a practised auscultator can distinguish the peculiar phenomena of cavities, in the snuffing, blowing, or tinkling and more articulate voice that certain spots present, or a coarser gurgling if liquid be present. You will readily perceive that all these phenomena are liable to be interrupted or modified by the accumulation of the matter secreted by the cavities and adjoining tubes. So also as, in time, the disease advances, the excavations become extended, and the gurgling first, and pectoriloquy afterwards, are heard in new spots.

You might at first suppose that the excavation of tubercles and the formation of cavities containing air, would remove the dulness on percussion, that accompanies the two first stages of phthisis: but this is seldom the case; for although there is more air than there was, yet there is much solid deposit about the walls of the cavities, and the irregular density and flaccidity of the parts, as well as the defect of air in the peripheral structure of the lung, still tend to check and muffle the vibrations of the walls of the chest, and prevent them

from yielding a clear sound. Even where the cavity is so large as to be the seat of a tinkling echo, or metallic tinkling, the resonance on percussion is irregular and imperfect; and thus may this case of metallic tinkling be distinguished from that of pneumothorax, in which some part of the chest must have an unnaturally clear sound. Sometimes the sound is clearer, in consequence of a general dilatation of the superficial cells; and as this is commonly of the flaccid kind, it may be accompanied by a sharp puerile kind of respiratory murmur: both these circumstances may disguise the phthisical signs, but only partially, for there will still be some decided irregularities in the sound on percussion, and enough of the signs of the subjacent caverns to declare the case to the wary observer. Now and then we find a case in which there is a hollow or bottle-like sound, on percussion over a cavity; this is when it is pretty large, and the intervening tissue is pretty uniformly condensed. More commonly there is an opposite condition; the walls of the cavity are loose and yielding, and, if it be large, percussion may sometimes cause a motion of its contents and a gurgling or tinkling expulsion of air from it, which produces a muffled metallic sound, like that of money in the nearly closed hands, or more nearly like the imitation of that noise with which we amuse children in striking the hands, hollow and closed, upon the knee. Laennec compared the sound to that emitted by a cracked jar when it is struck.

With the irregular and deficient sound on percussion, generally most obvious under the clavicles or in other parts of the upper regions of the chest, there is very commonly associated a collapse or sinking in of the walls of the chest, forming under the clavicles a remarkable hollow, generally more conspicuous on one side than on the other. There is very commonly, also, some defect and irregularity in the movements of the chest; the upper part of one side being but little raised, and the lower parts altogether exhibiting the most motion.—But there is seldom that complete fixing of one side that we see in chronic pleurisy, in which case, too, the upper part is generally more mobile than the lower.

We might class with the physical signs the characters of the sputa in the third stage of consumption, if they came only from the cavities which are peculiar to it. The expectoration of distinct portions of tubercle, or of pulmonary tissue, which have been seen in a few instances, constitutes a physical sign of the clearest character: they must come from cavities. If patients could save all their expectoration, and if this were inspected daily, this unequivocal sign might be more frequently met with. But the inflamed air-tubes are in great measure the source of the expectorated matter, which therefore presents much of the same aspect as in chronic bronchitis. The large size, and almost perfectly purulent character of the masses sometimes expectorated, which are like irregular balls of flock or wool, and of a yellowish or greenish colour, sinking and breaking down in water, go far to prove the existence of cavities in the lungs. A dirty yellowish brown or greenish matter, occasionally streaked or fringed

with blood, flattening like a piece of money when separate, and in masses forming a smooth sluggish purilage, are more characteristic of phthisis, and commonly occur in the most advanced stage. The general pulmonary congestion which frequently precedes death, is often announced by the darker reddish or green hue of the purulent sputa. Profuse hæmoptysis does not happen often in the advanced stage of consumption; for the vessels soon become plugged with fibrin and obliterated in the diseased portions of lung, and the mass of blood is reduced to the capacity of those that remain free.

I think I have said enough of the most distinguishing signs of consumption to make it unnecessary to pursue them further into the details of differential diagnosis, or the distinctions between particular diseases. There is only one kind of lesion which, even in its physical signs, may be mistaken for tuberculous excavations; I mean dilated bronchi. These, I have told you, may be the seat of a coarse gurgling ronehus, cavernous breathing, and pectoriloquy; and the accompanying chronic bronchitis often furnishes also a purulent expectoration. The situation, greater extent, and more stationary character of these lesions, may serve to distinguish them: they most commonly occupy the scapular, mammary, and lateral regions, and not the infra-clavian; they usually extend over a considerable space, and they do not tend to spread as tubercular cavities do. Again, if they arise from disease in the bronchi only, they do not impair the sound on percussion so much as phthisis does: and if they originate in pleuro-pneumonia, the dulness is much more complete, and is confined to one side, and accompanied by a more marked contraction than that which occurs in phthisis. But you should take the general symptoms also into account. There is seldom, with dilated bronchi, the degree of emaciation that occurs in phthisis; and when they arise from condensation of the lung, there is very often œdema and general dropsy, which are not common in simple phthisis.

It is not at all uncommon for phthisis to become complicated with other diseases of the chest; especially bronchitis, pneumonia, and pleurisy, and the attacks of these additional lesions sometimes prove fatal even when the phthisical changes are not extensive. Thus it is very common to find general pneumonia attacking a lung in which there are miliary tubercles, which must have existed prior to the inflammation, and would probably not have run their course for many months. This complication greatly increases the danger of pneumonia, which, unless it be stopped in its very onset, commonly proves fatal. In some instances, especially in the young, we see a reason of its intractability in the tuberculous character of the hepatization, which has the greyish or boiled-liver aspect, with considerable softening, instead of the redder deposit of common hepatization; on the pleura there is sometimes seen, at the same time, the friable opaque lymph which borders so closely on real tuberculous matter: and in other instances, where the disease has not advanced far enough to present these appearances, we can still understand that there may be in the deposit enough of that defect of vitality which renders tuber-

culous matter so difficult of absorption. The supervention of the signs of an extensive pneumonia, crepitation, with increasing dulness on percussion, affecting the posterior lobes of one or both lungs, together with the increased febrile disturbance, increased heat, and the rusty tinge in the sputa, must be looked on as indicative of extreme danger to patients with any extent of phthisical disease ; for if it do not itself prove fatal, as it very commonly does, the inflammatory attack will not fail to hasten and increase the phthisical disease. It is otherwise with the circumscribed pneumonia that not unfrequently attacks portions of the lung in the progress of tuberculous disease ; these come on without much disturbance, and subside without causing much mischief, being probably the result of mere local obstruction or irritation. The same remark applies to the slight pleuritic attacks so common in phthisis, the effects of which are seen in the adhesions of the pleura so generally found in phthisical subjects. I have often heard a sound of friction in different parts of the chest, continuing for some days ; then ceasing, and the chest when examined long after, has exhibited adhesions at these points. I believe that the inequalities occasioned by the deposits in the lung are sufficient to cause a local inflammation in the pleura, by their continual friction against it. Liquid effusions are less common, and arise from a more extensive cause, such as the bursting of a vomica into the pleura. If the vomica also communicate with the bronchi, there will be pneumothorax as well as liquid effusion. In either case the pleurisy is a serious and untractable addition to the consumptive disease, and may prove fatal in a few hours. Pulmonary hemorrhage is another grave accident, occurring especially in the early stages of phthisis. It may prove fatal by loss of blood, or by suffocation ; or the effusion of blood may break up the tissue of the lung to a great extent, and the patient may sink from the sloughy suppuration that ensues. Or, after the hemorrhage has ceased, inflammation may arise in and about the hemorrhagic consolidation, and involve the lung in a destructive suppuration, which may be more or less of a tuberculous character. On all these accounts it is of the greatest consequence to guard against or to arrest pulmonary hemorrhage, and when it has taken place to use means to promote the absorption of blood from the tissue, and to prevent the rising inflammation, which under these circumstances may prove so pernicious.

Destructive as phthisical lesions are, both by their own tendencies, and by the manner in which they affect the system, it is nevertheless now well ascertained that they do occasionally admit of cure. I think we are warranted in supposing that this may take place at any stage ; but the mode of cure which has been most completely traced, is that by the expectoration of the tuberculous matter, and the lining of the cavity with a complete false membrane, which commonly is of a fibrous or fibro-cartilaginous texture, tending to contract and ultimately to obliterate the cavity, but is sometimes thin and more like mucous membrane, without any obvious tendency to such a contraction. It is not uncommon to find in the lungs of those

who have long laboured under symptoms of pulmonary consumption, some of the cavities with a lining more or less perfect, and at the apex of the lung especially there may be now and then found a cavity contracted almost to obliteration; and sometimes a mere cicatrix perhaps inclosing a little friable caseous matter. All these instances evince a natural effort towards the healing of ulcerous cavities in the lungs; and where the disease is very limited in extent, and fresh tuberculous deposits do not take place in other parts, this healing of the cavities may amount to a cure of the consumption. The symptoms which may lead us to hope for such an unusual event, are a gradual diminution of the cough and purulent expectoration, a cessation of the fever, and quickness of pulse, and a decided improvement in flesh and strength. The signs that countenance this expectation are, the diminution of the pectoriloquy and cavernous respiration, and the restoration of some vesicular respiration, and a better sound on percussion in the part, whilst in the rest of the lungs the sounds are natural.

I cannot bring morbid anatomy to prove the possibility of a cure in the earlier stages of phthisis; but I have the history of several cases in which the signs render it extremely probable that some of the depositions which form the first stage of consumption of the lungs, had been removed by absorption. I have not time to give you the detail of these cases; but their general features are these: there have been cough, some shortness of breath, in two instances hæmoptysis, in some pain under the clavicle, quickened pulse, some evening fever; and these symptoms, instead of taking the course of an ordinary cold, have remained permanent for two months and upwards, with some loss of flesh and strength. On physical examination there have been found some dulness on percussion, on or under one of the clavicles or scapular ridges, with bronchial respiration, or a slight permanent ronchus, and increased resonance of the voice. Under treatment and favourable circumstances of air and climate, these symptoms and signs have been gradually removed, in some cases entirely, in others partially, but to such an extent that the patient's health has been considered in great degree restored. It may perhaps be said that we have no certain proof that these cases were phthisical, or if they were so, that the phthisical lesions were entirely removed. I admit this, but maintain that the existence of phthisis was as much proved as it ever can be in its early stage,—that precisely similar cases when neglected commonly run a consumptive career,—and we are not aware that such a combination of signs and symptoms can be produced but by lesions which essentially have a phthisical tendency.

Time will not permit me to dwell long on the treatment of pulmonary consumption, for if I were to enter into details they might well occupy two or three lectures. I must refer you to the recent work of Sir James Clark, which contains much important matter, especially on this subject; and I shall now give an outline of the best plan of treatment, in accordance with the views to which the study of the

disease has led us, with a few remarks suggested by my own experience on the effect of particular classes of remedial agents in the prevention and treatment of pulmonary consumption.

We have been led to conclude that the most important elements in the production of phthisical lesions, are a state of constitutional weakness, and a local vascular irritation or congestion : these elements predominate in various proportions in different cases, and will require a corresponding variation in the treatment ; but in almost every case both the constitutional and the local causes must be duly investigated and treated, or success will be only a matter of the most incalculable and irrational chance.

In the last lecture I divided cases of consumption into three classes : 1. Those originating from local disease ; 2. Those originating from constitutional disorder, or hereditary predisposition, without any previous local disease ; and 3. Those arising from local disease in a subject of hereditary or acquired scrofulous or phthisical constitution. I had occasion to observe, with regard to the first class, that the local disease might act, not only by developing in the lungs lesions which tend to run a phthisical course, but also by injuring the functions generally, and thus a constitutional cause becomes added to the local one. Therefore I believe in no case should we exclude constitutional treatment from a prominent place in the management of consumptive patients : and as far as a plan including many variations can be described by words, that best suited for consumptive cases generally may be designated as consisting in constitutional tonics, and local counter irritants or evacuates. It is, however, where local disease has been the chief cause of the mischief, that we have the best chance of curing consumption ; and the more so, in proportion as the local lesions are limited, and the constitutional powers little impaired.

The symptoms of the first stage of phthisis, that of the indurations, are those especially of vascular irritation, and obstruction ; and this is the period in which antiphlogistic and counter-irritant remedies avail most. Small general bleedings in the plethoric, and in those in whom there are symptoms of considerable congestion, or pulmonary hemorrhage, or repeated moderate leeching below the clavicles for the less robust, may often be practised with considerable advantage in the early stage of consumption ; especially when an increase of pain, cough, a bloody tinge in the sputa, dulness on percussion, and irregular respiration under the clavicles, indicate a congested state of the lung about the suspected indurations. In cases of greater debility, or where there appears to be a defect of blood in the system, blisters or counter-irritants are more suitable. I generally prefer a saturated solution of tartar emetic, rubbed in below the clavicles twice a day, until the skin begins to be elevated in little papulæ. If it be continued longer, the pimples may become pustules, and go on to suppuration, which is not desirable in this stage. When the papular eruption has died away, there being only a few pimples that have come to a head, the application may be renewed according as the symptoms may require. Issues and seatons commonly produce too much

irritation of the system to be useful at this stage. A more moderate and general counter-irritation may be produced by sponging or rubbing the whole chest once or twice a day with a stimulating liniment, such as the volatile liniment, or one composed of strong vinegar, with a little almond oil and some pungent volatile oil, such as that of cajeput or lemon.

The efficacy of internal sedative medicines is more doubtful, except so far as they tend to diminish the irritation of the cough and pain. Thus digitalis, colchicum, and hydrocyanic acid, may now and then subdue temporary vascular excitement, and thus quiet symptoms ; but I doubt the utility of continuing them long with the view to permanently reduce the pulse : they may thus do more damage to the constitution than they give relief to the irritations. The narcotic remedies, such as opium, conium, hyoscyamus, and belladonna, are also occasionally useful in allaying cough and pain, especially when these symptoms are associated with high nervous sensibility or a tendency to spasm ; but they have no permanent influence on phthisical lesions, nor on the congested vessels about them ; and unless given judiciously, they may disorder the gastric and alvine functions, and thus injure the state of the constitution.

But there are no remedies which will promote the removal of the indurations themselves ? Of this we can only speak doubtingly ; but if we may be guided by analogy, we might almost be led to hope that certain medicines may influence the process of absorption and renewal of the matter of textures, so as to facilitate the removal of morbid deposits. Thus we see tumors of various kinds, enlarged glands, and depositions in the joints, occasionally reduced under the influence of mercury, of alkalies, or of iodine ; and although there are many forms of deposit on which these remedies exercise no influence, and others in which their power is very equivocal, yet the limits of their action are not so well defined as to authorize us to say that all the kinds of induration which precede tuberculous deposit are quite beyond their reach. The influence of these remedies in promoting the absorption of the simpler products of acute inflammation is scarcely to be questioned ; and arising, as the lesions of phthisis occasionally do, from acute inflammation, and presenting various gradations which remove them only step by step from its products, it would be unreasonable to assert, without sufficient evidence to prove it, that these agents can never exert some such operation on the deposits that form the first stage of phthisical lesions. Evidence on the other side is also wanted ; and all that I can say on this point is, that in the cases of apparently arrested consumption to which I have before alluded, some of these remedies have formed part of the treatment. I cannot say much of *mercury* in such cases, except as an occasional aperient : it may, perhaps, be useful where the pulmonary lesion originates in acute inflammation ; but whatever its influence on this may be, its operation has generally appeared to me to be so injurious to the constitution, that I have been deterred from employing it to affect the

system in more than two or three instances : in these the disease was certainly not retarded by its use.

Nor can I speak from experience on the effect of frequent *antimonial emetics*, which were formerly much used in the early stage of phthisis, and have been lately again recommended on theoretical grounds. Before I can advise the use of so violent and disagreeable a remedy, I must have better testimony in its favour than that of old writers, who, we know, did not possess the means of distinguishing phthisis from chronic bronchitis.

In *iodine*, combined with alkalies, I rest more hope ; and I have been in the habit of giving this medicine in incipient cases of consumption for the last ten years. The form that generally agrees best is the hydriodate of potash, in doses of two or three grains, three times a day, with twenty or thirty minims of liquor potassæ, in decoction of sarsaparilla, infusion of calumbo, or distilled water, according to the state of the system, adding a little tincture of henbane, ipecacuanha wine, or any other medicine that the predominant symptoms may indicate, and always drinking or eating something farinaceous after it. Where there is a tendency to feverish irritation it may be given in a nitre draught ; and where there is more vascular debility it may be combined with mild tonic infusions. In chlorotic and in exsanguine scrofulous subjects, the iodide of iron is a suitable form ; and where this is borne, not causing headache and fever, or increase of cough, it rarely fails to improve the state of the general health ; but it should always be combined with occasional local depletion or external counter-irritation of the chest. When iodine agrees (and by varying its form and combinations it may generally be made to agree,) it increases all the secretions, and seems to give increased activity to the whole capillary system. In cases of gastric irritation, with pain in the stomach, or florid tipped tongue and thirst, it should be suspended, and a dose or two of hydrargyrum cum creta administered, followed by a few small daily doses of castor oil, or some other mild aperient ; and after a few days the hydriodate of potash may be resumed, guarded by the frequent use of a farinaceous diluent.

But even in the cases in which the phthisical lesions are most limited, we are never to forget that it is not these lesions alone that we hope to remove. Their very presence in the system, or the operation of the cause that produces them, may lead to the formation of more ; and in our treatment we should endeavor to remove those low degrees of vascular irritation, or that unhealthy condition of the nutrient matter of the blood, which, singly or combined, occasion the deposition of tuberculous indurations. We have already alluded to the local means of preventing vascular irritation and congestion of the lungs ; we must now advert to the constitutional treatment. Here we are to seek for all those circumstances and agents that may best promote the due action and balance of all the functions. The purest air and the most suitable climate for regular and ample exercise in it—the most nutritious food that the digestive organs can easily assimilate, and that the vascular system can bear without excitement—such re-

medial agents as give at once tone to the system and maintain the free action of all the secreting organs, together with friction, exercise, and proper clothing, to promote the activity of the superficial circulation—these are the means which are rationally indicated to fulfil the object of improvement of the general health. But how vague is this statement! how little can it guide us in particular cases! And so it must be: the means must be varied and adapted to the diversified forms of particular subjects, and it is in the study of individual cases, and in the power of discovering their conditions and of adapting means to them, that the ability of the practitioner is displayed. I can only give you the more general results of my observation in the employment of these various hygeian agents.

Pure country air is almost indispensable to give any chance to the consumptive. If the disease be limited and chronic, and his circumstances prevent him from giving up his employment in town, he should at least sleep in the country, and take every opportunity of longer absence. I have no hesitation in saying that I have known the lives of several consumptive patients prolonged by adopting this practice. But the country must be dry, and not too much exposed to the east and north, or there may be only a change of evils, from cachexia to inflammation. There is no air which is so truly an antidote to the poisonous effects of a town residence, as that of a dry sea coast; and the more open this is for the summer, and the milder and more sheltered in the winter, the better for the consumptive. The benefit that patients often quickly experience from the change is most striking, even in the more advanced stages of consumption. To profit fully by the influence of pure air, the patient should be as much out of doors as the weather will permit, and use as much gentle exercise, both by walking and riding on horseback, as the state of the strength will allow, without inducing much fatigue.

The diet in the early stage of consumption should be generally of a very mild unstimulating character: consisting chiefly of milky and farinaceous food. Sometimes white fish and chicken may be allowed; and a state of vascular debility, or previous habits in some cases make the plainer kinds of meat necessary; but this is especially the period of irritation and congestion, and more mischief is likely to result from repletion than from moderation. For the same reason, fermented liquors are seldom admissible at this period.

Of remedial measures, those already named in relation to the local lesions and particular symptoms, may be combined or modified so as to act favourably on the functions at large. This is especially the case with iodine. Occasional mercurial or saline aperients will be generally needed to prevent internal congestions, and to ensure the sufficient action of the abdominal viscera; but they should not be carried to excess, and their operation should be aided by due attention to the diet; so, also, the functions of the kidneys and the skin may, in particular cases, be ameliorated by aid of medicines; but the more that can be done by clothing, diet, and regimen in general, the better.—Clothing, especially, should be most carefully attended to; we have

in it the means of affecting, often powerfully, the whole vascular system ; and if so regulated as to maintain a permanently warm and supple, but not relaxed state of the whole surface and extremities, it would prevent many of those fresh colds and exacerbations that are the greatest bane of phthysical invalids. In case of these aggravations, which commonly consist in an increase of bronchitis, but sometimes of pneumonia or pleurisy, the remedies for these diseases must be cautiously resorted to ; always limited by the reflection that we are treating a subject already debilitated with disease, and in whom a permanent source of irritation will prevent that complete relief that antiphlogistic measures may give in simple inflammations. In case of hæmoptysis, much care is required to remove the congestion or vascular fulness, which occasioned it, before attempts be made to arrest it by styptics ; or the congestions may pass into inflammations, which, occurring in a lung tuberculated and consolidated by hemorrhage, is particularly destructive. Moderate repeated bleedings from the arm, or by cupping, and the use of tartar emetic in small doses, not sufficient to cause vomiting, joined with digitalis and nitre, a little morphia in case of vascular irritation, are the measures which I have found of most avail. If, in spite of these, the hæmoptysis continue to any amount, the superacetate of lead, in doses of two or three grains, with half a grain or less of the aqueous extract of opium, should be given every two hours, or as often as the urgency of the case may require.

In the second and third stage of phthisis, the chief modifications of the treatment are indicated by the greater degree of constitutional debility, whilst the symptoms of irritation may have diminished, or at least have not increased in proportion. Here depletions are less needed and worse borne ; and a somewhat tonic plan of treatment and more generous diet may sometimes be ventured on with advantage. Still counter-irritation generally proves useful ; and now that accompanied by purulent or sero-purulent discharge, will commonly produce the greatest benefit. In fact the same abatement of irritation that I have before described as accompanying free purulent expectoration, will in some degree follow from this external suppuration, without the wasting and harassing effect of such a discharge from the lungs. With this external outlet, as a sort of safety-valve, strengthening medicine and nourishment, may be borne ; and there is less risk in restraining any excessive secretion which may take place from the lungs, the bowels, or the skin. Much attention is necessary to keep up the discharge, whether it be by the formation of successive crops of pustules, by tartar emetic solution, or some similar suppurating liniment, or by a seton or issue. If it be suddenly checked there will, in all probability, be an increase of pulmonary irritation, probably attended by the deposition of more tuberculous matter. In slighter cases, or where the weakness or irritability forbids these measures, occasional blisters, or the frequent use of milder liniments, containing tartar emetic and hydriodate of potass, or acetic acid and oil of turpentine, are often productive of some benefit.

In the tuberculous and ulcerative stages of phthisis, the constitu-

tional powers especially need support ; and it is then, more particularly even than in the first stage, that the general measures are required. But unless the disease be limited in extent, there is, for the same reason, less hope of their success. The disease has existed longer, and passed into a stage in which it is more likely to have tainted the system. The preparations of iodine, and other alteratives, should be used more freely, and the general health supported by all the medicinal and hygeian circumstances that can be brought to bear on it. There are vomicæ to be evacuated, and the object is to get them emptied, and to promote the healing of the fistulous cavities which they leave. Can this be aided by local applications, by inhalations of chlorine, iodine, or other vapours ? Some who have used these means speak highly of them ; too highly to gain our confidence. I have seen them used, and do much harm ; but I do not deny that, under very judicious management, they may sometimes contribute to induce a healthy healing action in the interior of ulcerated lungs. But we must look more to an improved state of the constitution for such a healthy action, and for what is of more immediate moment, a cessation of that disposition to deposit more tuberculous matter in other parts, which too commonly prevails during the softening and evacuation of the tubercles.

In very many cases, alas ! no means will stay the progress of consumptive disease ; and the utmost that we can do is to give temporary relief to troublesome symptoms ; to the cough, by various forms of opiates ; to pains in the side, by a blister or a mustard poultice ; to the dyspnœa, by æther and ammonia, or by the tincture of lobelia ; to the hectic heats, by sponging with vinegar ; to the sweats, and to excessive expectoration, by acid mixtures ; to the diarrhœa, by astringents, preceded by a mercurial aperient ; and so forth. But in not a very small number of cases we may considerably prolong life by watchfulness and care. Consumption may run its course in a few weeks ; but it may exist in a limited and chronic form for many years ; and it is these long cases that may reward us for our attention and judicious treatment, if not by permanent recovery, at least by temporary restoration of a moderate share of health and strength compatible with the enjoyment of life, and the fulfilment of important duties in society.

I have occupied so much time with the subject of phthisis (without, however, having half exhausted it,) that we have scarcely any left for that of encephaloid, scirrhus, melanose, and similar productions, affecting the lungs. Their occurrence is too rare to be of much practical importance ; and they are not known to be in any degree influenced by medicine. They may occur in a circumscribed form, or occupying a considerable extent of the texture ; and they would then produce physical signs like those of consolidation from hepatization or tuberculation of similar extent, and could be distinguished from these only by the history and general symptoms, and by the absence of the constitutional indications of tubercles. They commonly cause death, either by their encroachment on the func-

tion of the lungs, or from being simultaneously deposited in other organs, such as the mesentery, the liver, the ovaries, &c. But when they occupy the lung chiefly, both encephaloid and melanose deposits tend in time to soften and form ulcerous cavities as in the case of tubercle. I have seen such cavities more than once in both these forms of disease. I have not met with a sufficient number of cases of these morbid deposits to enable me to give you their anatomical history, as I have that of tubercle. The general appearance of *encephaloid disease*, or *medullary sarcoma*, is that of a brain-white solid, of varying consistence, with a pinker hue than that of tubercle, occurring either in separate tumors, which are sometimes encysted, or infiltrated through the tissues of the lung, and modified by their colours. When occurring in separate tumors it is sometimes soft and cellular; in other cases tougher, and more like the pancreas; in others again, as in this specimen, it becomes of fibro-cartilaginous hardness. A predominance of a loose cellular and vascular structure in it, with patches of extravasated blood, give it occasionally the appearance that has obtained for it the name of fungus hæmatodes. We may conjecture that the albuminous matrix of these products is deposited in an organizable form, and vascular ramifications are certainly formed through it; but it is deficient in the cohesion and contractile tendency of ordinary false membranes; it does not restrain the further effusion from the vessels, whence the tendency to growth in these productions. When encephaloid matter occurs in an infiltrated form in the tissue of the lung, it sometimes presents an appearance intermediate between that of tuberculous and that of hepatized consolidations; and unless there be portions of the diseased production occurring separately, it might be taken for one or other of these lesions.

The only form of disease which I have seen affecting the lung, which approaches in any degree to *scirrhus*, is that which I have already described as a result of a chronic pleuro-pneumonia; there being in these cases firm adhesions to the pleura, a shrunk state of the lung, and dilatation of the bronchial tubes. The induration and glistening texture which the lung so changed sometimes exhibits, especially around the larger air-tubes, might lead one to suspect it to be of a scirrhus nature; but I have seen neither the tendency to cancerous ulceration, nor the simultaneous occurrence of scirrhus in other parts, which might be expected if this induration of the lung were really of a malignant kind.

I have met with several cases of *melanosis*, or *black tubercle*, affecting the lung, both exclusively, and with the same production in other parts of the system. In this drawing you see it combined with encephaloid disease. The black matter may occur infiltrated in a natural structure, or in distinct tumors or deposits of an irregular cellular organization. I am much inclined to adopt the opinion of Andral, that the black matter is nothing but a modification of the colouring matter of the blood, in which carbon is in excess, or even in a free state. In the lung from which this drawing was taken,

the deposits exhibited in different parts various shades of colour, from the dark cruor red of hemorrhagic engorgement to the deep jet black of perfect melanosis. The intermediate colours were of a bistre or sepia brown. The organized texture of melanose tubercles and tumors presents considerable variety, sometimes approaching to the most perfect products of acute inflammation, being soft and cellular or membranous; and sometimes having almost the totally unorganized structure of scrofulous tubercle. Probably it is only this latter form that undergoes the changes of softening and ulceration ascribed to melanosis by Laennec; and under these circumstances, such changes are to be referred to the same causes as those which operate in the kindred changes of tubercle. The presence and modification of the colouring matter of the blood seems, therefore, to be the essential pathological condition of this disease, as an altered or deficient vitality of the fibrinous matter is of tuberculous affections.

It is necessary to guard you against confounding with melanosis the accumulations of the black pulmonary matter, which take place to a great extent in the lungs of old people, especially among the inhabitants of large towns. These are probably, as Dr. Pearson supposed, derived from the soot inhaled with the air; which, I presume, finds access to the texture of the lungs chiefly through abrasions, softening, or other such lesions of the bronchial membrane, which, in a slight degree, often result from a common cold or cough. Whether from this source, or, as others have supposed, from an altered state of the colouring matter of the blood itself, I think it is plain enough, that when once deposited in any corners out of the immediate sweep of the circulation, such as in the angles of lobules, near old lesions, around large vessels, and in the bronchial glands, there it must lie, accumulating until death, or until it is carried off by the destruction of the tissue by some pulmonary disease. For it consists entirely of carbon; and this being totally insoluble in any animal fluid, is insusceptible of absorption, which cannot act on insoluble solid matter. For the same reason the carbonaceous matter of tattooed skins, and the insoluble oxide or chloride of silver in persons coloured blue from the too long internal use of nitrate of silver, are permanent, and can only be removed with the skin itself. It does not appear that the carbonaceous deposit in general interferes materially with the function of the lungs; but there are some curious cases on record, in which it has taken place so rapidly and extensively as to cause chronic inflammation and consolidation of a perfectly black colour, which tends to ulceration and the formation of cavities, as in other cases of chronic consolidations. Such cases are described by Drs. Gregory, W. Thomson, and others, as occurring particularly in coal-mines, and in persons labouring under bronchial disease whilst continually employed by the light of smoky lamps.

The general symptoms of encephaloid or melanotic consolidation of the lungs, are those of obstructed breathing or circulation, dysp-

nœa, lividity, and dropsy ; more commonly than those of consumption and emaciation, which belong rather to tuberculous disease. This is explained by their more rapid development, and their not so readily leading to softening and ulcerative destruction of the organ. When this process does occur, the expectorated matter may afford means of distinction. I have seen, in the case of encephaloid disease, streaky red and white purilaginous liquid sputa, and, in melanosis, a considerable quantity of black matter, mixed with a muco-purulent compound. But such cases are not common, and the expectoration is more usually that of the bronchitis or pneumonia that may accompany the disease.

The *bronchial glands* are not unfrequently found after death in a diseased state, even when no symptoms referrible to them had been manifested during life. I do not mean the deposit of black matter like that of the lungs, for that is so constant that it can scarcely be considered to be morbid. But they are sometimes found swollen and red, or containing caseous matter, or osseous concretions. In children they are occasionally so enlarged by the deposition of tuberculous matter, as to press on the air and blood-vessels, and, according to Dr. Carswell, to produce dyspnœa and symptoms of obstructed circulation. They sometimes soften, and become evacuated by ulceration into the bronchi. Encephaloid disease, as well as extensive enlargement, not of a malignant character, may also affect these glands. I suspect that encephaloid disease of the lungs generally originates in this way, and spreads afterwards along the vessels into the pulmonary tissues. I have also met with cases of dulness on percussion on the top of the sternum, with signs of obstructed circulation and respiration, with simultaneous enlargement of the axillary and cervical glands, betokening a glandular tumor about the root of the lungs ; and all these symptoms gradually subsided under the use of iodine and alkalies ; so I conclude that the tumor was simple glandular enlargement. Considerable tumors of the bronchial glands might perhaps sometimes be discovered by dulness on percussion on the upper portion of the space between the clavicles, and on the spinous processes of the upper dorsal vertebræ. The tumors, generally, however, grow forwards, and I have seen them pushing out the sternum or the ribs on one side, and causing dulness at those parts, and symptoms of displacement of the lung further down. They also may produce signs by their pressure on the great vessels, arterial and venous, as you will understand when we treat of the organs of the circulation—a subject which I propose to begin in the next lecture.

LECTURE XXIV.

On the Heart, its Structure and Mechanism.—Its Position.—Its Vital Properties and Motions.—Physical Examination of the Heart.—By Feeling, Impulse, Varieties; by Percussion; by Auscultation.—Description of the Sounds of the Heart.—Causes of the Sounds.—First Sound.—Second Sound.

HAVING endeavoured to give you a connected and intelligible view of the physiology and diseases of the organs of respiration, I shall now, as fully as our time will permit, explain to you in the same manner the natural and diseased properties of the other organ of the chest—the *heart, and its appendages*. Four or five years ago I could not have conscientiously undertaken this task, for I found so much in the phenomena of this organ, in health and disease, inexplicable and inconsistent with the views and descriptions of even the most recent writers, that the subject was on every side beset with difficulties and obscurities, which defied all attempts to exhibit it in a rational manner. Since that time, however, it has been investigated extensively in different quarters; and having myself devoted much of my time to it, I think that the physiology and pathology of the heart now admit of the same rational and connected exposition which we have attempted with regard to the other organs of the chest.

The heart, you know, is a compound hollow muscular organ, consisting of its four compartments, lined inside with a serous membrane, and enveloped outside by a proper fibrous capsule, besides a serous covering, which is a part of the pleura, whilst another fold of the same membrane forms the external pericardium or sac; this has also beneath it a fibrous layer, which seems to be continuous with the cervical fascia. Now, that we may understand how the heart contracts, let us see what is the origin and insertion of its fibres. It is not difficult to trace these after boiling the heart for a long time, which dissolves the gelatin of the connecting cellular membrane, whilst it hardens the fibres. By this means it has been ascertained that the greater number of the fleshy bundles arise from, and are inserted into the strong fibrous rings which form the mouths of the great arteries, and similar rings which form the auriculo-ventricular openings, or into tendinous prolongations from them. Some of these run obliquely downwards, and around both ventricles, and are inserted into opposite parts of the same rings. Others pass around the left ventricle only; and the outermost and longest run obliquely towards and around the apex, and then passing into it form the fleshy lacework of the interior, and the columns to which the tendinous cords are attached. There are a considerable number of fibres, especially near the base of the heart, which encircle the ventricles, especially the left, without any definite origin or termination; these,

by their contraction, tend more directly to diminish the transverse circumference of the ventricles, as the longer fibres diminish their length by drawing the apex towards the base. The auricles are formed more simply of loops variously crossing each other attached chiefly to the tendinous rings which open into the ventricles. The contraction, then, of all these muscular fibres, is towards the auricular and arterial orifices, which are the most fixed parts of the the organ, and the effect of the contraction must be to press the contents of the cavities towards these openings. And it is thus that we see the heart act, on opening the chest in animals that have been deprived of sensation by a narcotic poison, or by a severe injury to the nervous system. Attached by the vessels at its base, and with its apex free, it is drawn together towards these vessels at each contraction; and the anterior surface being more convex, from the fibres being much longer than those behind, their contraction is greater, and the apex is also drawn forwards as well as upwards.



Now let us glance at the valvular mechanism of the heart, by means of which its contractions propel the blood in a determinate

course. In order to show this well we must be careful how we cut open the heart, or we may injure the apparatus, and render its mode of action indistinct. [See the engraving.] I first make an incision in the left ventricle, from the apex close to the anterior groove, which marks the position of the septum, to the mouth of the aorta, taking care to cut between and not across the semilunar valves. This incision displays the arterial valves, and the arterial portion of the ventricle. But to see the auricular portion and mitral valve, I cut again from the apex, at right angles with the first incision or with the plane of the septum, up to the auricular ring; and thus you have both laminæ of the mitral valve fully displayed *in situ*, without severing one of its tendinous cords. It is well to remember this mode of opening the heart; for if it is useful in showing the natural structure, it is especially so in tracing the changes of disease in it; and I have often seen lesions overlooked, and fine morbid specimens destroyed, in consequence of the heart being opened at random.

The office of the semilunar valves is most obvious, from their mere mechanical structure. Attached by the whole of their convex ventricular margins, they fall loose and unresisting against the sides of the arteries at each gush from the ventricles; but no sooner does this gush cease, and the distended arterial column press backwards, than their loose arterial margins are caught by the first turn of the reflux current, and they are distended into three sacs, the free sides of which being in close contact, completely intercept the passage of blood back into the ventricles. This action is merely mechanical, and can be produced in the dead body; it will be more perfect in proportion as the backward pressure from the arteries is greater. The auriculo-ventricular valves, on the other hand, will not act well after death: their office depends on the vital contraction of the fleshy columns, to which their cords are attached, as well as on the mechanical spread of their laminæ. You see that these muscular pillars draw down both laminæ of the valve, but they cannot bring them together with any force so as to close them. This is done by the lateral pressure of the blood, which, directed by the contracting ventricle on both laminæ, closes them one against the other, and effectually prevents regurgitation into the auricle.

The pulmonary or arterial portion of the right ventricle may be displayed in the same way as the corresponding portion of the left; but the different position of the tricuspid requires that the second incision should be made from the middle of the other cut [see engraving] instead of from the apex, and carried round to the posterior groove. You here see the three irregular triangular curtains of the tricuspid valve, which are drawn in succession one somewhat behind its neighbour, but all more across than in the axis of the ventricle, as in the case of the mitral, so that the valves close the orifice more in the manner of the semilunar valves, but still under the varying vital influence of the fleshy columns, which, by contracting more or less, may complete, or leave imperfect, the closure of the valves. Hence when the right ventricle is much distended,

the curtains of the tricuspid valve do not entirely reach across the orifice, and regurgitation takes place. This seems to be a provision against an excess of pressure on the pulmonary vessels, and induced Mr. Adams, of Dublin, first to call this valve a safty valve. The subject has been lately well illustrated by Mr. T. King.

The auricles may be opened by a long crucial incision, which displays their interior, their ventricular and venous orifices, and their septum, in which is the semilunar groove of the foramen ovale. There are some other points in the anatomy of the heart that are worth adverting to ; such as the roughness of the auricular and the smoothness of the arterial portions of the ventricles, and the remarkable adaptation of the structure for the transmission of the blood in one direction. We have no time to dwell on these ; but I must particularly commend to your attention the anatomy of the human heart. It is an organ easily examined, and by attention to the directions above given, you may soon acquire such a familiarity with its common appearances, as may better qualify you to appreciate the change induced by disease, and without this familiarity no description, however minute, can suffice.

The heart, you know, is placed in the anterior mediastinum, rather to the left of the mesial line, and so oblique that the apex points forwards and downwards to the left, while the base lies back nearer the posterior centre, the spine. It therefore lies with its point on the diaphragm, underneath which are the liver and stomach ; and it is bounded on other sides by the lung, except a small space of about two square inches, where, enveloped in its coverings, it is in contact with the walls of the chest.

Little need be said here about the *vital properties* of the heart. Its contractions are essentially periodic and involuntary, and perhaps independent of the nervous system ; but this is a subject much debated : they are certainly, however, liable to be influenced in a variety of ways, both by the nervous system and by the blood. Any sudden impression on the nervous system may stop the heart's action, or it may accelerate it. Thus, crushing the brain or spinal marrow will stop it, and smaller injuries may quicken it ; but both the brain and spinal marrow may be removed without either of these effects. In fact, the influence of these injuries to the nervous system does not appear to be different from that of injuries to other considerable parts of the body : thus, Dr. M. Hall found that crushing a limb had the same effect of arresting for a time the heart's action. I do not think that we should attach much importance to such difficult and equivocal experiments as those recently made by M. Brachet, who describes the heart's action to cease instantly on cutting the great cardiac plexus. If you only recollect the anatomical position of this plexus, and how impossible it is to reach it in a living animal without extraordinary disturbance of many vital parts, you may well question the conclusiveness of a result so obtained.

The influence of the blood in exciting the contractions of the

heart is more general than that of the nervous system, and there can be no doubt that it is the proper stimulus of this organ, exciting it by both its quantity and quality as it fills its cavities. At the same time we must not forget that the rhythmical contractions of the heart will continue for a short time when it contains no blood, when it is taken out of the body ; and if we call to mind, besides, the numberless instances of disease in which the heart's action may be permanently increased both in force and frequency by causes which do not affect the quantity or quality of the blood, we must admit that there is some cause of the property and its varieties, independent of the blood which is its ordinary excitant. Whether this be a *vis insita* or a *vis nervosa* cannot, in the present state of our knowledge, be determined.

Let us now see what we can learn by the physical exploration of the *heart in action* ; and first, by the sense of *feeling*. On applying the hand to the left front of the chest, between the fourth and sixth ribs, the pulsations of the heart may be distinctly felt ; but they vary remarkably both in strength and extent, according to the stage of the respiratory act and the position of the body, as well as from differences in their own strength. In a well-formed chest, the impulse of a healthy heart, is scarcely, if at all, perceptible when the individual takes a full breath, or lies on his back. On making a full expiration, on the other hand, or on stooping forward, especially to the left side, it becomes strong, and is spread over a considerable surface, being felt much higher than usual. You see, then, that the position of the heart is not fixed in relation to the walls of the chest, but that it hangs to a certain degree loose, and liable to displacement by change of posture and by the motions of the chest. The common position, however, is such that only the apex is felt beating at a very circumscribed spot between the cartilages of the fifth and sixth ribs ; in males, commonly about two inches below, and in front of, the left mamilla, and about the same distance from the left margin of the sternum. A deep inspiration elevates the ribs without raising the heart in the same degree ; hence it makes the apex beat below the sixth rib ; the impulse is then, however, diffused, and scarcely perceptible, because the chest expands as the ribs rise, leaving the heart, and drawing the porous lung in front of it. A forced expiration, on the other hand, depresses the ribs, and transfers the strongest pulsation to between the fourth and fifth ribs, and by bringing down the walls into contact with more of the heart, makes its impulse perceptible over an extended space, as high as the third rib and on the lower half of the sternum. All these circumstances, although wholly unnoticed by Laennec and other writers, are of great importance, and should be known, not only to guard us against undue comparisons, but also to furnish us with the means of testing the free state of the heart and lungs, and of bringing the power more within reach of our examinations. Whilst you feel for the impulse of the heart, you should therefore desire the patient to vary his posture, by leaning forward on your

hand, and first to take in and then to give out a full breath. The natural impulse will, for the same reasons, vary according to the form of the chest and the extent to which the lungs are developed in front of the heart. In narrow or distorted chests, and in those contracted after pleurisy, the impulse of the heart may be felt much more extensively than usual. So also circumstances displacing the heart, such as tumours, and effusions of liquid or air into the pleura, may greatly change the character and degree of the impulse, diminishing or increasing it according to whether the displacement of the organ is from or to the walls of the chest. Abdominal tumors, and even a distended stomach, may to a certain degree have a similar effect. You are to remember also that the impulse or perceptible motion of the heart is naturally intercepted or circumscribed by the soft porous tissue of the air-filled lung, and that it may be therefore modified by changes in that tissue. Hence pulmonary emphysema may still further intercept or circumscribe the impulse: consolidation of the lung, on the other hand, will propagate it over an extended space. The greater thickness and strength of the left ventricle make its motions more forcible and extensive than those of the right; and it is perhaps for this reason that this ventricle is placed to act chiefly on the soft cushion of the lung, which offers no unpleasant resistance to it. You see by the position of the heart that the apex is almost the only part of the left ventricle that comes far enough forward to strike the walls of the chest. The front is constituted by the right ventricle which lies under and even a little to the left of the sternum, and it is chiefly the impulse of this ventricle that is felt there on a full expiration, or on leaning forwards. It is by the comparative degree of impulse to be felt there, and further to the left, where the apex strikes, that Dr. Chambers chiefly judges of the relative condition of the two sides of the heart.

The examination of the region of the heart by *percussion* should be immediately added to that by feeling, and will be found to afford satisfactory illustrations of its results. In a well-constituted chest of a person who is not fat, there is commonly some dulness on percussion from the left margin of the sternum to the extent of between one and two square inches towards where the impulse is felt; but this dulness is remarkably diminished, if not quite removed, by a leaning back or supine posture, and by taking a full inspiration; and it is as notably increased by leaning forwards and to the left, and by a forced expiration. This is, then, another method of testing the free condition of the heart and of the lungs that involve it. For example, if such variations are not observed—if in common states of respiration, and especially if on full inspiration, or on leaning back, there is a considerable extent of dulness on percussion at and to the left of the sternum—it may be concluded either that the heart, from adhesions or from its bulk, cannot recede from the walls of the chest, in which case the impulse would also be increased; or that there is considerable effusion in the pericardium, in which case the impulse would be diminished. But we shall come

to these applications afterwards ; I wish you now to understand the principles on which this mode of examination indicates the condition of the heart. To define the limits of the heart by percussion, we have to attend, not only to the *perfect dulness*, which exists where the organ is in absolute contact with the walls of the chest, and which, in disease, sometimes extends over an area four or five inches in diameter ; but we must notice also the *shading off* of this dulness, where the lung overlaps the organ. To discover this we must use forcible mediate percussion, which gives the sound of the deeper-seated contents. By this means we may sometimes trace an enlarged heart when very little of it comes in contact with the walls of the chest. In emphysema of the lungs the permanently distended state of the anterior lobes nearly intercepts all impulse, and makes the region of the heart quite resonant ; but strong percussion may still indicate that there is an enlarged dense organ beneath this resonant surface. In considerable enlargement, the sound of strong percussion on the left back, and in the left lateral region, is also somewhat impaired.

We now come to consider the examination of the *acting heart* by the sense of *hearing*. I shall first describe to you the chief facts of the subject, and afterwards enter into an explanation of those facts. If you listen either with the stethoscope or with the ear alone applied to the region of the heart, you hear at each pulse two sounds following each other in quick but regular succession, and succeeded by an interval of silence until the next pulsation. The first is a long, rather dull sound ; the second is a short abrupt flap. Some French writers have used a very wrong sounding word for the double sound, by calling it a *tic-tac*. If it is to be given at all by the symbols of articulate sounds, the word *lubb dup* will best express it. If we would only be a little more consistent in our rules of pronunciation, I think we might often give the sounds of the heart, healthy and morbid, in language of this kind, with more ease and precision than by descriptions or comparisons. It is a very convenient mode of expression in taking notes of cases, and I probably shall have to use it in describing some of the morbid sounds. Laennec endeavoured to give expression to these sounds, by marking their rhythm or measure. Supposing the period of a pulse (that is, the time from the commencement of one double sound to the commencement of another) to be divided into four equal parts, he described two of these as occupied by the first sound, a little more than one by the second sound, and the remaining less than one by the interval of silence. These sounds present much variety, so that it is difficult to estimate their proper length ; but I should note the average rhythm somewhat differently from that given by Laennec, who makes the second sound longer and the interval shorter than what I have generally heard. I will represent the measures by a diagram, which will be more generally intelligible than musical notation. The uppermost is that given by Laennec, the other is what appears to me to be the more usual rhythm in health.



This is the general character of the sounds in the healthy male adult. In females and in children the first sound is rather shorter and less dull, more like the second ; and the same difference may be observed when the pulsations of the heart are frequent or weak.

The physical causes of these natural sounds of the heart have been the subject of much discussion. Laennec's opinion was, that the first sound was caused by the contraction of the ventricles, and the second by the contraction of the auricles ; but he did not advance any proofs in support of this opinion, nor did he attempt to explain how these respective contractions could generate the sounds. The late Professor Turner, of Edinburgh, was the first to test the matter by physiological experiment ; and he was thus led at once to detect the error of Laennec, by observing (what had been before noticed by Harvey and Haller) that the contraction of the auricles immediately precedes that of the ventricles, and therefore cannot cause the second sound. This observation of Professor Turner was afterwards confirmed by the experiments of Dr. Hope, in which it was proved that the first sound distinctly accompanies the contraction of the ventricles, and that the second sound occurs at the moment of their diastole, and is in no way connected with the motions of the auricles, which appear to be too slight to cause any sound. But the immediate physical causes of both sounds were undetermined until February, 1835, when, assisted by Dr. Hope and others, I conducted a course of experiments in the room below, which led to pretty conclusive and satisfactory results. These experiments were afterwards repeated and varied by the Dublin Committee of the British Association for the Advancement of Science ; and more recently an extended series of experimental researches on the same subject has been conducted by Drs. Clendinning, Todd, and myself, in another Committee for the same Association. You may find the different reports of these investigations in the *MEDICAL GAZETTE* ; they have pretty thoroughly sifted the subject, and it is satisfactory to me to be able to say that they confirm, in all material points, the results and conclusions of my first experiments, which you will see fully given in the third edition of my little work on the Pathology and Diagnosis of Diseases of the Chest. We have not time to describe these experiments, nor indeed, is it necessary. If we consider the structure and action of the heart, we shall be rationally led to perceive how it may produce the sounds, and I will merely adduce the experiments to guide and answer the questions which arise from this examination of the acting mechanism.

It was decided unequivocally, by all the series of experiments alluded to, that the first sound accompanies the whole duration of

the systole of the ventricles, which also causes the impulse felt on the walls of the chest ; it was equally clear that the second, or flapping sound, occurs at the first moment of the diastole, and that the motion of the auricles was not accompanied by any perceptible sound.

Then, first, how does the contraction of the ventricles produce the first sound ? Sound we formerly defined to be *motion of a certain force resisted with a certain force* ; where is the resisted motion in the contraction of the ventricles ? One of the first ideas that suggest themselves is that of Magendie : does not the heart produce the impulse by striking against the walls of the chest ? and why should this not cause sound ? I answer, that in forcible pulsations, and when the lung does not intervene, I have no doubt that the impulse does produce sound ; and if you listen to the sound of the heart when it is beating strongly, or when, by leaning forward or by breathing out, the heart is brought in contact with the walls of the chest, you will hear the first sound has something like a knock in it, which you can scarcely help referring to the impulse. But this is an accessory, and not an essential sound ; for you may hear the first sound when there is no impulse, as in a person leaning backwards, or taking a full breath ; and in our experiments we heard the first sound at the origin of the arteries when the body of the heart was surrounded with the soft lung or with tow, or was allowed to hang out of the chest, and strike against nothing in its motions. It is well known, too, that liquid in the pericardium, or liquid or air in the pleura, although it entirely prevent the heart from striking the walls of the chest, does not annul the first sound. In moderate pulsations the heart makes a partial rotatory movement ; the long fibres of its anterior convex surface drawing the apex upwards and forwards, and causing it to slide obliquely on the smooth pericardium, bring it to the walls of the chest too gently to produce sound, except under the circumstances before mentioned.

The first sound is, then, produced by something in the heart itself, either by its contents or by its own structure. Can it be by its contents ? by the motion of the blood resisted by the inequalities of the interior of the ventricles ? This was supposed by the Dublin Committee to be the chief cause of the sound. But observe, that the motion of the blood over these inequalities is not rapid, nor is their resistance considerable at the time of the production of the sound. These inequalities are, as Dr. Cowan has pointed out, chiefly confined to the lower or auricular portions of the ventricle, into which the blood has already passed before the systole begins ; and the effect of the systole is to drive the blood, not over or across the inequalities, but from them, to those smooth and funnel-shaped parts of the ventricle which lead to the arteries, and which offer the least possible resistance to its course. The contraction of the ventricles propels the passive mass of blood gradually, that is, during the whole period of the first sound, into the arteries ; and the motion is therefore too slow and easy to be likely to cause sound.

Some of our experiments further settled this point, by proving that the first sound continued when the ventricles contracted without any blood in them.

By excluding the blood we are thus brought to the conclusion that the cause of the sound must be in the solid structure of the ventricles ; it is our next question whether it be in any part of them in particular. Several writers have ascribed it to the auriculo-ventricular valves, which, when they close, are supposed to produce a flapping sound. But the act of closing these valves is momentary, and takes place only at the commencement of the systole ; whereas the first sound of the heart is prolonged through its whole duration. Further, in some of our experiments the first sound continued, although impaired, when the auriculo-ventricular valves were prevented from acting, by fingers introduced into their orifices, or by some of their cords being cut. Still these valves may produce a part of the sound, for at each contraction they are suddenly tightened in a manner calculated to generate sound.

But are the valves the only parts which are tightened at each systole of the ventricles ? Is not every muscular fibre in the ventricles suddenly tightened by this action ? Here are the elements of sound, motion vigorous and rapid, suddenly resisted by the mass of blood to be urged forwards by the contraction, and the contracting motion and the resistance, although greatest at first, continue to act as vibrating forces during the whole systole ; hence the prolongation of the sound. In other instances, abrupt and forcible muscular contraction produces a sound like the first sound of the heart. Apply the stethoscope to the adductor muscle of the thumb of your closed hand, and contract the muscle strongly and quickly. Or, to avoid the possibility of the joints being the seat of the sound, apply the end of a flexible tube to the abdominal muscles, and start them into sudden vigorous action : you will get sounds quite as loud as those of the ventricles, and very like them in character. By varying the mode of muscular action you may get different kinds of sound. When the contraction is slow or sustained, however strong, you get only the dull rumbling noise which Dr. Wollaston first described, and which he attributed to a vibration depending on a regular intermittence in the force of the contraction. When the contraction is gentle and slow it may cause no sound at all ; as we have seen that the auricles produce no sound, neither do the ventricles, when their contraction is very feeble. But whenever there is strong abrupt muscular action in any part of the body like that of the heart, there will be heard a sound which will resemble that of the ventricular systole, in proportion as the muscles in which it is produced resemble in thickness and density the tissue of the heart. The loudness of the sound is by no means in proportion to the thickness or strength of the muscle, but rather to its simplicity, and the abruptness as well as the vigour of its contractions : the transition of a thick muscle from slack to tight can never be so complete and sudden as that of a thin one ; where there are many fibres they choke or muffle each other's vibrations ; hence the sound is dull and pro-

longed rather than loud and clear. Many writers who have objected to my explanation of the first sound of the heart, have done so in ignorance of the principles on which muscular action causes sound ; when these are known the identity of the phenomena becomes apparent ; and in my experiments there was the best proof that we could have that the muscular contraction of the heart produced systolic sound, for we had the heart out of the body, without its blood, without valvular action, lying on the table, or on my hand, and its contractions were still accompanied with a sound, weak indeed, but in character resembling its natural first sound.

The walls of the ventricles appear to be peculiarly calculated to generate sound ; their flaccid state when relaxed, the fineness of their fibres, and the harmony with which they suddenly contract on their contents, and become almost as hard as a stone (as we can feel in the living heart of a stunned animal) fulfil the conditions best calculated for the production of sound. The commencement of the systole producing the tightening of the auricular valves, and thereby completing the resistance of the body of blood on which the contracting fibres have to act, is naturally its loudest part, and often has a flapping character ; that which continues after is more dull, and is prolonged according to the quantity of blood to be expelled, and the continued strength of the contraction. This prolonged termination of the sound is, therefore, best heard when the heart acts slowly and vigorously.

And now what causes the second sound ? That it is intrinsic, and not caused, as Magendie supposed, by the heart striking any of the surrounding parts, we proved by the same experiments, in which the intrinsic character of the first sound was shown ; the sound continued when the heart was so completely isolated that it could strike against nothing. What is there, then, within the heart that can produce this short flapping sound at the moment of the diastole ? Is there anything that tightens at that moment ? Not the walls of the ventricles certainly, nor the auricular valves, for they are then all loose and flaccid. What can it be but the semilunar valves at the mouths of the arteries, which are then suddenly tightened by the reaction of the arterial column of blood ? And so it was proved to be, in my experiments first, and in many repeated since ; for by hooking back these valves, or by pressure preventing the reaction of the column of blood upon them, the sound was stopped ; and by releasing the valves, or discontinuing the pressure, the sound was as surely restored.*

So much for the causes of the natural sounds. We shall consider their variations in the next lecture.

* According to M. Bouillaud, Dr. Carswell was the first who conceived that the second sound is caused by the flapping of the semilunar valves : but I cannot find that the opinion was made public. The first proposal of this explanation that I know of in print, is in a thesis by Dr. Elliot (*De Corde*), Edinburgh 1831. M. Rouanet, Dr. Billing, and Mr. Carlile, were later advocates of this view.

LECTURE XXV.

Physical examination of the Heart (continued).—Analysis of the Causes of the Sounds.—Varieties of the first Sound; from the contracting Walls; from the Quantity of the Blood; from the Action of the Valves; from the impulse against the Ribs, &c.—Varieties of the Second Sound.—Distinction of the two Sounds.—Abnormal Sounds or Murmurs.—Acoustic Explanations of their Production, with illustrations.—Pathological Causes of their Varieties.—On the Arterial Pulse as a Sign of the Action of the Heart.—Signs of a simultaneous Examination of the Heart and Pulse.—Varieties of Pulse from Diseases of the Heart.

CONSIDERING it, then, established that the first sound of the heart is produced essentially by the tightening of the valves and walls of the ventricles by muscular contraction, and that the second sound is caused by the sudden tension of the arterial valves by the recoil of the arterial column of blood upon them, we proceed to examine how far these sounds may represent the condition of the parts which respectively produce them; how they may be modified by changes in these parts, and by the addition of new elements of sound arising from such changes.

The elements of the first sound are the contraction of the walls of the ventricles, and the resistance given to their contraction by the blood within them. Each of these elements may vary in character and give corresponding varieties in the nature of the sound. You can see at once that force and briskness in the contraction must together increase the sound, and that briskness gives it loudness, but not duration, which is rather caused by strength and continuity of action. Hence a heart that is contracting abruptly may generate a loud sound, even when its contractions are not strong; and a heart acting strongly, but slowly, will produce a duller sound, and of longer continuance. These different modes of contraction are frequently dependent on the thickness of the walls of the ventricles; but not constantly. You can readily understand why the contraction of a thin muscle should be abrupt and short, from its simplicity and want of mass: and why a thick muscle should be slower, although stronger in motion. Observe the different sounds produced on tightening this thin silk and this thick baize. The thinness of the silk gives a unity and briefness to the impulse which it receives, and the sound is short and loud. In the baize the impulse is divided and prolonged in the complexity of the fibres, and the sound is dull and less brief. So a thin ventricle will give a louder sharper sound than a thick one, under similar circumstances. But when under the influence of increased irritability, a thick ventricle contracts with more abruptness than strength, it may give as short and as loud a sound as a thin one which is acting slowly and with more tone. I have frequently heard a hypertrophied heart, when excited by nervous irritation, produce sounds louder than those of a dilated heart that was acting

quietly. In this case the stronger impulse from the thicker heart would still distinguish it ; but there are some conditions in which even this test may fail ; for instance, during or after syncope, the action of the heart is abrupt and feeble ; and even when the walls are thick, the sound is generally short or flapping, and the impulse weak. It would seem that the systolic action is incomplete or partial, confined perhaps to the outer fibres and the tension of the valves being abrupt enough to be sonorous, but insufficient to cause an impulse. These variations, which are exceptions to the rules given by Laennec and other writers, are explained by the view which we take of the production of the sound, and ought to be known, or they may lead to error. Although the ventricles are two, yet their fibres are in great part continuous, and their action and sound is one. But the different thickness of the two causes the sound to be shorter and clearer at the sternum which is over the right ventricle, and duller and longer where the apex of the left ventricle beats between the fifth and sixth ribs ; and these differences are more remarkable where, in consequence of disease, the difference in thickness is augmented.

The other element contributing to the production of the first sound is the resistance opposed to the contracting ventricles by the mass of blood within them. Now the amount of this resistance will depend on the quantity of the blood, and the opposition that it meets with in its passage from the ventricles. When the quantity of blood is great, it will resist the ventricular contraction longer, and therefore maintain the sonorous tension longer than when it is small, but the resistance will not be so sudden, and the sound not so loud : for if the heart be distended with blood before the systole begins, its fibres cannot be so loose, and the great cause of sound, the transition from slack to tight, will be less complete. On the other hand, when there is little blood in the ventricles, they pass from great flaccidity to tension so suddenly that the sound may be unusually loud, but it can have no continuance. As far as the blood is concerned, therefore, a dull prolonged sound is a sign of a large quantity, and a short loud one of a small quantity, expelled from the ventricle. In accordance with this we find that when the pulse is slow, other things being equal, the sound is much longer than when it is quick ; and such a length of sound in a pulse that is not slow, and the heart not diseased, becomes a sign of sthenic plethora. The longest first sound that I ever heard was in a gentleman whose pulse beat only twenty-eight in a minute ; it occupied nearly a second of time. But this is not the case with all slow pulses, for where the pulse is slow from weakness, as in syncope, its contractions may be brief and incomplete, and the sounds short and weak. A short sound with a slow pulse is essentially a sign of weakness, and this may occur in asthenic plethora or cardiac congestion. When there is little blood in the system, we find the sound remarkably short and flapping, and it may be very loud ; but

then the pulsations are commonly frequent, and the radial pulse small.

But besides the quantity of blood, the resistance to its expulsion from the ventricle may modify the character of the first sound. If the vent for the blood be free, the resistance will be brief, and the sound short. Now the chief resistance opposed to the blood is that of the semilunar valves tightened by the pressure of the arterial column, and this will be great in proportion to the fulness of this column; here is then another reason why an abundance of blood tends to lengthen the systolic sound; there is more blood to be expelled, and more resistance to its expulsion. On the other hand, when there is little blood in the arteries, the systole takes place suddenly with little resistance, with a short sound, and throws the blood into them in a loose sudden jerk, which is often felt in the pulse of those who have lost much blood. A permanent narrowing of the arterial orifice by disease must also tend to lengthen the first sound by prolonging the systole; but as such a change develops a new sound or murmur at the constricted point, it will be better to notice it afterwards. There is, however, one more element in the resistance given to the systolic contraction which must be noticed as a chief cause of the commencement of the sound—that is, the closure of the auriculo-ventricular valves. Until this takes place, the resistance scarcely begins, for the passage back into the auricle is too free; but no sooner have these valves closed, than the ventricles tighten on the ball of blood, and the sound is produced. You can see then how the free perfect play of these valves contributes to the first sound, not only by their own tightening, but also by their completing the strain of the other walls of the ventricles. The closure takes place suddenly, almost at the beginning of the systole, and thus causes the loud and flapping commencement of the sound. Where from disease their closure is not sudden or complete, this flapping commencement is less perceptible. There may besides be a new sound or murmur produced by the current of blood through the unclosed valve, but this we shall consider by and by.

You see that in the production of the first sound many elements are concerned, and although the process is simple enough in itself, you must be fundamentally acquainted with its parts before you can fully understand the varieties of sound, and safely interpret them as signs. I suspect that very few will attempt to do this; the majority will content themselves with the easier but far less exact rules given by Laennec and others. I consider myself bound to describe the phenomena as I have found them, and to explain them as far as they will bear it; but not to sacrifice accuracy to simplicity. I have not done yet with the varieties of the first sound; for besides those of duration and loudness there are other differences in its character.

Sometimes the systolic sound, without being longer than usual, seems broken into two by something like a flap in the middle; or to enunciate it, instead of being *lubb-dup* as usual, the first and se-

cond sounds are *bullub-dup*. I am doubtful as to the cause of this variety, but I think it probable that it may depend on some irregularity in the action of the auricular valves, especially the mitral, by which their tightening with its attending flap is either delayed or takes place in two successive jerks. This kind of sound occurs most commonly in cases of diseased heart; but I have heard it sometimes where there was no reason to suspect permanent lesion, but where the action of the heart was languid. In these cases the impulse likewise is sometimes double, and from the same cause. A double impulse is also now and then produced by the action of the valves following with a jerk the first stroke of the apex against the ribs; this, too, may make the first sound double.

The systolic sound has sometimes a remarkable drum-like character, without any murmur or other irregularity, the note of the heart being then *lumb-dup*, or *lung-dup*. Dr. Chambers, who has also heard this sound, described it as *gong-like*. As far as I can remember, this sound has been observed only in cases where there were signs of liquid in the pericardium or left pleura; and if this be the case, we may explain its more musical character by the contractions of the ventricles taking place in a medium which leaves their vibrations more free than usual, not muffled by a closely-wrapped membrane and the spongy lung. In these cases the impulse is diminished, and this separates them from those with the metallic clink which frequently accompanies the sounds of a strongly-acting heart, and which Laennec referred to air in the pericardium, whilst recent French writers have been imagining other causes equally fanciful. This metallic clink originates in the ear of the observer, and has nothing to do with the heart further than that it is excited by the blow which the heart communicates to the ear. You may hear the very same note if you lay your ear flat upon the table, and tap the under side of the table just opposite to it. I believe this to be the proper note of the tympanum. You may often hear it in auscultation, but when once aware of it, you can easily recognise it as belonging to yourself, and not to the patient. It is especially produced when the heart strikes against the ribs, and the external knocking sound is added to the intrinsic systolic sound. The addition of the knocking to the first sound is very perceptible when the heart beats strongly, or in the circumstances of posture, state of the respiration, and shape of the chest, before mentioned as most favourable to increase the impulsion of the heart against the ribs; but it is so much incorporated with the intrinsic sound that it seems to form a part of it. This is not surprising when we consider that the sounds produced by at the same time striking and straining a body, must partake of the same character, and become incorporated, seeing that its tension and mode of vibration are for the moment uniform. The sound is no doubt partly generated also in the walls of the chest; and when the impulse is very abrupt, it gives the impression of a knocking sound.

Besides the intrinsic sound and that of impulse which is thus

occasionally added to it, the heart's motions sometimes produce sounds in the adjoining parts. The friction sound of the pericardium I shall notice hereafter ; but beyond this, a strong impulse of the heart on a portion of lung may forcibly press the air from it ; and if there happen to be any partial obstruction or mucus in its tubes, a short sibilant or mucous rhonchus may accompany each beat. The character of these additions, and the circumstance that they accompany the breathing also, and are more or less diminished by holding the breath, or by posture altering the manner in which the heart beats on the lung, may serve to distinguish them from the true cardiac sounds. Again, I have repeatedly heard the tinkling echo of pneumo-thorax—of a large cavern—of dilated bronchi—nay, even of an inflated stomach, accompany the pulsations of the heart when excited ; these circumstances may be easily detected by their other signs, as displayed by auscultation and percussion.

The *second sound* of the heart is more simple in its character and causes, and less liable than the first sound to be modified by circumstances. Caused solely by the sudden reaction of the arterial columns of blood on the semilunar valves, its loudness will depend on the mobile and perfect state of these valves, and the extent and abruptness with which they are stretched by the recoil of the blood at the moment of the ventricular diastole. It will, therefore, be most perfect when the heart acts regularly and slowly, giving time for a full gush of blood to carry the valves loose into their slight recesses in the walls of the artery, and for the as perfect reaction of the contents of the distended artery on their concave surfaces. On the other hand, if, from the quickness or weakness of the ventricular contractions, the jets of blood thrown into the arteries be small, the play of the valves will be less complete, and the sound less distinct. Also, if the permanent distension of the arteries be great in proportion to the propulsive power of the heart, as it sometimes happens during fits of palpitation, the valves may not in many pulsations be opened enough to flap distinctly. Or if the arterial tension be greatly diminished, as under the immediate influence of profuse hemorrhage, it may not close them with sufficient force to give a sound. The second sound is generally heard most plainly at about the middle of the sternum, or between the third and fourth ribs close to it, which is opposite to the situation of the arterial valves ; but it is generally audible over most of the upper parts and front of the chest ; in the normal state perhaps more extensively than the first sound. Although there are two sets of valves, aortic and pulmonary, yet being brought into play at the moment of the diastole, which is simultaneous in both ventricles, they are generally tightened at the same instant, and produce but one sound. But if from the less mobile state of one set of valves, or from the greater pressure of the blood on them in one artery, the tightening of the two sets do not coincide, the second sound may be double, consisting of two flaps in quick succession, so that the note of the heart is then *lubb-dupup*, or *lubb-durrup*. This sound sometimes closely

resembles in rhythm the footsteps of a cantering horse. It rarely occurs (never, I believe, for a continuance) in cases where the heart is free from disease. When the action of the mitral valve is also retarded in the first sound, both sounds may have this double character.

I must not conceal from you, that it requires some practice in auscultation to enable you to distinguish some of these varieties of sound ; and if the action of the heart is irregular or quick, it is not always easy to distinguish between the first and second sounds. When the impulse against the ribs is pretty distinct, this being felt either by the ear, or by the finger applied at the same time as the ear, will sufficiently declare its companion the first sound. When the impulse is obscure, the finger may be applied to the pulse in the carotids, which is not perceptibly after it. The radial pulse is so much later that it may not unfrequently even accompany the second sound. The different characters of the sounds will often serve to distinguish them ; and when these are little marked in the region of the heart, they are often quite obvious at the top of the sternum, or in the carotid arteries, where the flap of the second sound can seldom be mistaken. The sounds in these arteries are, under ordinary circumstances, merely those propagated from the heart ; but, under the influence of violent action, I have no doubt that a sound is produced by the shock of the blood against the sides of the great arteries, and is heard over them as a loud short sound, as intense as the simultaneous first sound of the heart, but more abrupt. So much for the natural sounds and the variations from them, all of which are seated in the solids of the heart and its appendages.

We have argued by reasoning, and have shown by many experiments (see the investigations before quoted), that although sounds accompany the motions of the solids, yet the motion of the blood through the heart and vessels is habitually accomplished with the smallest possible resistance, and therefore wants one element essential to generate sound. But under various circumstances of disease this element, resistance, may be produced in the obstructions to the current of the blood, or in the novel channels into which it is thrown ; and thus are developed new sounds, which, as distinct from the natural sounds, we may, with Dr. Forbes, class under the general term, *murmurs*. An example or two will set before us something of the character and causes of these murmurs.

At the moment when the blood is expelled from the ventricle, if, instead of a smooth, equally tense channel, it meets with a rigid constriction, or an abrupt orifice, its passage through it will be attended with a whizzing or blowing noise, which may be heard in the region of the heart. Or to take a simple case : if you apply the stethoscope in the course of an artery far from the heart, you will hear nothing as long as the current of blood flows smoothly and unmodified through it ; but if by pressure you diminish the caliber of the tube at any point, you then complete the elements of sound ; you give resistance to the moving blood, and at each pulse

you hear a blowing or a whizzing sound, which will vary in tone and loudness according to the force of the current and the degree of resistance which it meets with. This is purely a physical phenomenon ; you may produce it in any tube through which a current of water runs. Thus, if you take an India-rubber tube, and adapt it to a reservoir of water, so that the water shall flow freely through the tube, you may, by pressure on the tube, produce murmurs, varying according to the force of the current and the resistance which the pressure opposes to it. They are sometimes like blowing ; sometimes like rasping or sawing a piece of wood ; and now and then they may be heard in quite a musical tone, which implies that the vibrations are then regular and sustained. The blowing and musical murmurs are generally caused by greater regularity but less force of current than that which produces the sawing or whizzing sounds.*

In fact, all these murmurs are produced by the passage of *liquids* through solid tubes or apertures in the same manner as analogous sounds are produced by the passage of *air* through pipes or holes of different kinds. They are the music of *water-instruments*, as the latter are the music of wind-instruments. There are only these differences between them, that liquids being more sluggish than air, are less susceptible of the sudden motions which constitute sonorous vibration, and not differing so much in density from the solids in which they move, liquids will have little of those reflected or echoed vibrations which increase and modify the sounds produced in air-filled tubes. Holding in mind these qualifications, we may explain the murmurs heard in the heart and arteries by referring to parallel instances of the tones of wind-instruments ; nay, we may find the parallel phenomena in the rhonchi, respiratory, and vocal sounds of that most complete and diversified wind-instrument, the wind-pipe and its branches. Like in these, there are varieties of sound, in generating which the solids and the current have different shares ; thus in the grating, sawing and stronger droning murmurs, the vibrating resistance of the solid is chiefly concerned ; and its vibrations are transmitted to the adjoining parts as well as to the current, so as to produce in them a thrill which may sometimes be felt by the hand.—Being in the rhythm of the heart's motion, this thrill resembles that felt on the back of a purring cat, whence Laennec called it the "*frémissement cataire*." These have their parallels in the sonorous rhonchus, in reed instruments, and in those imitations of these murmurs which we can produce by forcibly breathing through the nearly closed teeth, tongue, and lips, which in like manner commu-

* These statements have been since amply confirmed by numerous experiments performed by Dr. Todd and myself, in committee, for the meeting of the British Association for the Advancement of Science, held at Liverpool last year. The report is published in the *MEDICAL GAZETTE* for December 2, 1837. In these experiments every variety of cardiac and vascular murmur was imitated, by variously modifying currents of water through caoutchouc tubes of different sizes and shapes, and the sources of variety were in great measure determined.

nicate a sensible vibration to the solids, as in ringing the letter *R*, in a whisper. Again, in the blowing, hissing, whistling, and cooing murmurs, the vibrations are more those of the current reflected by the solid, in the manner of cavernous breathing, the sibilant rhonchus, blowing, or whistling with the mouth, or of the flute class of musical instruments. Here there are no perceptible vibrations in the solids; they are less actively concerned in the production of these sounds, which are rather transmitted in the direction of the current.

Although I adhere to this view (which I first proposed in 1828) as the only one which gives a complete physical explanation of all cardiac and vascular murmurs, I must not omit to tell you that many other explanations have been proposed. Most of these are too vaguely stated to be really explanatory, being mere references to the indefinite influence of friction or obstruction. There is one, however, which is more precise, and none is more plausible or ably supported; it is that of Dr. Corrigan, of Dublin, to whom we owe many interesting observations on the pathology and diagnosis of diseases of the heart and arteries.

Dr. Corrigan considers that murmurs are produced not in the constricted or resisted portion of the current, but in the flaccid walls beyond, which are thrown into vibration by the rippling motion which the current assumes there. This view, he thinks, explains the occurrence of murmurs in the uterine arteries and in varicose aneurism, where, instead of constriction, there is an unusually free passage for the current; but from the changed calibre of the vessels, it is of a rippling kind. Dr. C. has illustrated this view by a number of ingenious experiments, for an account of which I must refer you to his paper in the Dublin Medical Journal. I think, however, that this view of Dr. Corrigan is partial, and mistakes an effect for a cause. The flaccid state of the tube beyond a constricted point is an effect of the impediment which the constriction opposes to the current, and the rippling motion of the current equally originates in the constriction, although being once produced it continues beyond it in the looser part of the tube. To prove this, cut off this flaccid part, and let the tube terminate with the constricted point, you will still have the murmur produced in it. You can try this experiment after lecture, with a current of water through this elastic tube, or with this India-rubber bottle, from which, by sudden compression, you can produce a current like that from the heart. You will find, that if you oppose a resistance to the current, either by pressing on the tube, or mouth of the bottle, or by holding something in it, the murmur is produced as well at the terminal orifice as at any other point. In these cases the constricted portion is like the mouth-piece of a wind-instrument, it alone produces the sound; but when so produced, the tube beyond may receive and modify it.

How then, you will ask, are we to explain the murmur in the uterine arteries and in varicose aneurism, in which there seems to be no constriction? I would refer you to the parallel case of a wind-instrument, the flute, in which also there is no constriction; for after

the wind has left the lips, there is free vent enough for it, and yet the sound may be of the loudest kind. There may be a vibrating resistance to the current without constriction: for instance, when the current enters a new and enlarged channel, at a considerable angle, and strikes against its sides, it communicates to them an impulse which they resist, and the series of impulses and resistances which thus ensue, if forcible and rapid enough, constitutes sonorous vibrations. The dilated and tortuous state of the arteries of the gravid uterus, and the unusual direction of the arterial current into a lateral or a varicose aneurism, presents conditions well calculated to produce sounds in this manner.

I will now briefly state the chief pathological conditions causing different kinds of murmurs, which may prove signs of those conditions:—

1. Constriction or projection in the arterial orifices; causing a murmur with the first sound, generally rasping or whizzing: very common on the left side of the heart.

2. Constriction or impediment in the auriculo-ventricular orifices, causing a murmur with the second sound: very rare.

3. Imperfect closure of the arterial valves, causing a murmur from regurgitation, with or instead of the second sound, generally whiffing or grating, sometimes musical: very common in the aortic valves.

4. Imperfect closure of the auriculo-ventricular valves, causing a murmur from regurgitation, with, and often impairing, the first sound: generally blowing, sometimes whistling or cooing: very common on the left side.

5. Considerable dilatation or relaxation of the aorta or pulmonary artery, just above its orifice, which, although of normal dimensions, is narrow, compared to the tube beyond it; causing a murmur with the first sound, generally grating: rather rare.

6. Partial obstruction or projection in a blood-vessel; causing a murmur with the pulse; blowing, musical, whizzing, or grating; prolonged in proportion to the degree of obstruction: common.

7. Abrupt aneurismal dilatation of an artery; causing a murmur with the pulse; generally grating; rather common.

6. Unnatural opening from a ventricle, or from an artery into another cavity, sac, or vessel, causing a murmur with the pulse, generally whizzing or rasping; if from the ventricle it must terminate with the second sound; if from the artery it may continue beyond it; rare.

It may be generally said of all these murmurs that they are increased by augmented force of the heart's action, and they may sometimes be changed by this influence; the blowing becoming whizzing or rasping, the musical murmur raised in pitch, and the like. So also the constriction of an orifice or artery, the defect of a valve, or any of the other causes may be present in so low a degree as to produce sound only under the temporary influence of an excited heart. On the other hand, I have met with instances, but they are very rare, in which increased action has diminished or even removed

a murmur. The quantity of blood in either extreme may also modify these sounds : when excessive, it increases and prolongs them ; when very defective, and accompanied by an excited action of the heart, it may make them loud and short. Even the quality of the blood may influence the sounds ; for a thin watery fluid is more readily thrown into sonorous vibrations than one of a richer, more viscid character ; and this is one reason why murmurs are so easily produced in chlorotic and anemic subjects. In these the slightest pressure of the stethoscope on the carotid arteries in the neck is enough to cause a loud continuous, or remittent whirring, to which the French have given the name of "*bruit de diable*," from its resemblance to the noise of a toy called a "*diabie*."*

It is in similar conditions that a murmur strongly generated at the orifice, or in the course of an artery, may be propagated through a great extent of the tube beyond, being carried with the current, which retains the vibrations, and may even communicate them to the finger by a sensible thrill. Sometimes the thrill is perceptible without the murmur ; this takes place when the vibrations are too slow to produce sound. It gives the feeling of something rough passing rapidly under the finger, and is quite distinct from the hard throbbing pulse of arteries leading to an inflamed part.†

*By experiments performed by Dr. Todd and myself since these lectures were delivered I have been convinced that this and other continuous murmurs, like the buzzing of a fly, heard in the neck, are more commonly produced in the jugular veins. Dr. Ogier Ward had previously discovered these to be their seats. They may be arrested or diminished by pressing on the vein above, by hanging down the head, or by efforts to expire with the glottis closed ; and they return with increased loudness on interrupting these acts.—(See Report of Committee, &c. MEDICAL GAZ., Dec. 2, 1837.) These sounds are not essentially morbid ; for they may be produced by the pressure of the stethoscope in the healthiest individuals ; but in accordance with what I have stated in my lectures, they may probably be more readily produced when the blood is thin and deficient in quantity. The production of a sound by a partially obstructed venous current gives us a new physical sign, and its continuous character makes it in a great measure distinctive of its seat. I have lately met with several cases in which its presence assisted me in the diagnosis of tumors within the chest. It is commonly a continuous humming or droning sound, heard near the clavicle or top of the sternum, or between the scapulæ, diminished by efforts at expiration, and increased immediately after them, or by contrary efforts.

† Professor Graves, in one of his interesting communications on inflammation (MEDICAL GAZETTE, July 7, p. 606), has objected to my explanation of the throbbing hard pulse of arteries leading to an inflamed part. I attribute this phenomenon chiefly to their dilatation, and to the obstruction of the vessels in the inflamed parts, to which they lead. That they are dilated has been proved by the experiments of Drs. Alison, Thomson and others—(See Fourth Report of the British Association for the Advancement of Science, *ibid.*, p. 674). That the passage of blood through an inflamed part is more or less obstructed when the inflammation is established, seems to be proved by the observations of Hunter, Thomson, Philip, Hastings, Gendrin, and others, and is in accordance with the known effects of inflammation. In ascribing the throbbing of the vessels to these causes, I merely connect it with known conditions, which seem to me to explain it. But Dr. Graves thinks that they do not explain it. He observes, "*the dilatation of the vessels, however caused, can on no principle account for their becoming the seat of throbbing and a hard pulse ; their being more open than*

I do not believe that either increased or diminished action is capable of producing a murmur in healthily constituted hearts and arteries. There must be something to modify, as well as to hasten the current, before it can produce sound. Neither do I see it possible that an altered shape of the ventricle can cause a murmur in the arterial orifice, without either constriction or projection in that orifice; or, what is equivalent to it, dilatation or relaxation of the artery beyond it. In fevers, inflammations, and nervous irritations, we have every degree of increased action; but no murmur, without other disease; and I have met with hearts enlarged and dilated in various ways, but producing no murmur, without some change in the orifices or arteries. This statement is not in accordance with the opinions of other writers; but it is given as the result of my experience and study, and it increases the value of murmurs as diagnostic signs, if by their characters or positions they can be traced to their mechanical causes. That in many cases they can be so traced, I hope to show in the following lectures.

The *arterial pulse* is another sign by which we judge of the action of the heart. I need scarcely commend it to your attention; for it has been too long and too universally consulted to be in danger of neglect. It is seldom so correct an index of the action of the heart as the sounds and impulse of the organ itself; yet some of those impracticable sages, who find physical signs in general too troublesome to be studied or valued, have exhausted their ingenuity with refined distinctions in this one physical sign, thereby only giving proof that there is more sense in their fingers than in their heads. I have formerly had occasion to explain to you the elements and the chief varieties of the pulse. (See Lecture XI.) Depending as it does on the size and condition of the arterial tube, and on the quantity and quality of the blood, as well as on the motions of the heart, it may be varied by many circumstances that do not affect that organ, and the motions of that organ may be variously intercepted or modified before they reach the artery, which is commonly felt by our fingers. But although these circumstances impair the accuracy of

others to the pulse-wave from the heart could at the utmost only place them in the situation of other arteries naturally of the size they have now attained to; but we do not find that such arteries throb, or have a hard pulse." Dr. Graves surely cannot mean that a small artery dilated, and with its terminations more or less obstructed, is in the same circumstance as a large artery undilated, and with its terminations free. The coats of the latter have that share of elastic strength that equalizes in every artery the natural pulse. The coats of the former being already made tense and thin by dilatation, receive untempered each pulse from the heart, which here consequently becomes hard and throbbing. "Arteries do not throb, or become the seat of a hard pulse in proportion to their natural size:" but arteries do throb when their size is increased at the expense of their elasticity, and when opened to an impulse from the heart, which is unnaturally great. I had no time nor inclination to discuss in my lectures the theory of inflammation, which I leave to the more acute powers of Professor Graves; and in explaining the principles of the leading varieties of pulse, my aim was to exhibit the relation of well-ascertained phenomena, and to render them rationally instructive in diagnosis and practice.

the positive indications of the pulse with regard to the heart, if we only take the trouble to analyze them, they may give us useful information, positive or negative, with regard to the condition of the circulation.

The radial pulse in general represents truly the number of the heart's contractions ; it never can exceed them ; but when the heart acts very feebly, its pulsations may not reach the wrist ; and when they are irregular in force, some may be propagated to it and others not, in which case the pulse intermits. On listening to the heart, the ineffective pulsations are heard, and they are often loud enough, but their character, together with the fact that they do not reach the wrist, may inform us as to the morbid condition of the organ on which they depend ; and we may learn from the same examination, that however violent the heart may be seeming to act, it is defective in its power to propel the blood through the system. This inconsistency between its apparent and its effective power is often manifest on comparing the pulsations of the heart itself during a fit of violent palpitation with the comparatively weak pulse that reaches the wrist. These discrepancies do not, however, occur in every case ; for in some individuals the radial pulse pretty precisely represents the number, force, and even the time of the contractions of the ventricles, however varying they may be. This it does in those individuals in whom the coats of the arteries are rigid and unyielding, and transmit unmodified each pulse-wave from the heart. Where, on the other hand, the arteries are thin, elastic, and imperfectly distended, they may soften the hardness of the heart's pulse, in its course through them, reduce its strength, retard its period, and if it be small, annul it altogether. Thus, with your ear at the heart, and your finger at the same time on the radial pulse, you may take a far more accurate survey of the condition of the circulation than you can by examining these parts separately ; and the utility of this method is by no means confined to affections of the heart. Thus, in inflammatory fevers, the pulses at the wrist owe their hardness and strength in some measure to arterial tone or tension, for they are more instantaneously propagated from the heart, and *accompany* rather than follow the ventricular systole. In debilitated atonic states of the system, on the other hand, the radial pulse *follows* the first sound of the heart by a distinct interval, which is occupied by the transmission of the wave along the course of the comparatively lax and yielding artery, and it is in passing through such a tube that the pulses lose also their strength and hardness, and those that are weak may be altogether suppressed. Thus, independently of disease of the heart, a pulse that reaches the wrist tardily and weakly, is a sign of an atonic or asthenic state of the vascular system ; whilst one that is transferred with celerity and force, equally indicates that fulness and tension of the vessels which is the chief character of sthenic or inflammatory conditions. I recommend this comparative examination to you as well worthy of your attention, for I have often found it, as well as the character of the sounds of the heart, give positive practical indi-

cations in various diseases, when the pulse alone and other symptoms wore a doubtful aspect.

The character of the pulse may be very much modified by diseases permanently affecting the vital properties or mechanism of the heart and arteries. These varieties will be noticed as we treat of these diseases. I shall here only exemplify some of the different elements which are concerned in their production. A large strong heart acting with a prolonged first sound will cause a strong full pulse, which will be hard in proportion to the tension of the arterial coats. A large weak heart with a short first sound will produce a pulse which may be sharp or abrupt, but not strong, and it generally occupies an appreciable interval of time in passing to the extreme arteries. It may be small or large, according to the size of the artery and the general fulness of the vessels. Defects in the valvular apparatus of the left ventricle tend also to modify the pulse by altering the manner in which the blood is propelled from it. When the mitral valve does not close perfectly during the systole, part of the strength of this action must be lost backwards into the auricle, and the pulse will be weakened in proportion. This regurgitation commonly produces a murmur, and where it is considerable the pulse is also generally irregular. When the aortic valves are imperfect, the tension of the arteries is not maintained beyond the period of the ventricular systole; for when this ceases, the blood returns into the ventricle with a freedom proportioned to the insufficiency of the valves. This occasions a very peculiar character in the pulse. Owing to an increased thickness of the walls of the ventricle which commonly accompanies this lesion, the pulse is strong at the moment of the systole, but recedes instantly after, from the absence of the arterial tension, so that it has a jarring or jerking character which is very remarkable, and which often makes its motion visible, the artery starting into a slightly tortuous line at each pulse. This visible pulsation of the arteries has been described by Dr. Corrigan, as a sign of incapacity of the aortic valves. Constriction of the aortic orifice diminishes the strength of the pulse, but, unless extreme, not its hardness and sharpness. Constriction of the auricular orifice is generally accompanied with great irregularity of the pulse.

LECTURE XXVI.

Functional Disorders of the Heart.—Increased or inordinate Action.—Causes with Illustrations.—Pathological Effects of Increased Action.—General Symptoms; Palpitation.—Physical Signs of increased Action.—Irregular Action of the Heart.—Irregularities in Rhythm; in Strength.—Signs and Causes of Irregularity.—Defective Action of the Heart.—Syncope; its Causes.—Symptoms and Signs of Syncope.—Habitual Weakness of the Heart.—Its Causes and Effects.—Increased Sensibility of the Heart.—Phenomena and Causes of Angina Pectoris.

HAVING said as much as our time would allow, on the heart, its vital and physical properties and phenomena, and the modes of examining

them, we now come to its special pathology—to trace the modes in which its functions and structures may be affected by particular kinds of disease, and the phenomena that it may present under these affections. In doing this, we need not adopt the same order as in diseases of the organs of respiration. These organs are more complicated, and their functions are constituted by so many parts (the nervous system, the vascular system and its blood, and the mechanism of respiration), that their functional diseases are often less simple than the inflammatory, which, therefore, we first considered. The function of the heart, however, is so complete in itself, and its mode of action so determinate, that we may well introduce its diseases by a notice of the disorders of its function, which may be independent of inflammation or change of structure, although these are not unfrequently their causes.

Functional disorders of the heart may be divided into those of *increased* action, those of *irregular*, and those of *defective* action.

The action of the heart may be increased by three classes of causes.

1. An undue irritation by its proper stimulus, the blood.
2. Extraneous irritations acting either mechanically or through the nerves.
3. Increased irritability of the heart itself.

1. The blood may become unusually stimulating, from its quantity or from its quality. The increased and quickened action of the heart resulting from exercise is caused by the greater quantity of blood that is returned to the organ in a given time, chiefly by the pressure of the muscles on the veins. This acceleration ceases as soon as the blood returns only in its usual quantity. But the increased quantity may be permanent; as when there is an excess of blood in the system; the increased action may then be either in frequency or in force, or in both, and is reducible by blood-letting. But this plethora may be only local, and confined to the heart and other internal organs; as when, from a contraction of the superficial vessels under the influence of cold or sudden mental emotion, the blood is accumulated inwardly; or as when pressure on the great vessels, impeded respiration, or other cause of obstructed circulation, interferes with the flow of blood from the heart. Any of these circumstances may excite the heart to inordinate action, which may begin suddenly, continue until they be relieved, and even afterwards, if the heart be in itself irritable, and cease either suddenly or gradually. Again there can be no doubt that the quality of the blood may, under some circumstances, become unusually stimulating. The injection of diluted ammonia or spirit into the veins of animals is immediately followed by an increase of the heart's action; and it is reasonable to suppose that a similar effect from the ingestion of stimulating liquids is in part owing to the same cause. It is highly probable, too, that the accelerated pulse of several constitutional affections in which the blood is known to be altered, such as gout, rheumatism, urinary disorders, and certain fevers, is, in part at least, due to the stimulating quality of the blood.

2. It is very common for the heart to be excited by extraneous

causes. The pressure of an effusion or tumour in the chest, a distended stomach, or other abdominal tumor, may quicken its action by their mechanical irritation. But the more frequent irritations are those of sympathy through the nerves, especially the par vagum and great sympathetic. Through these channels strong mental emotions, acidity, flatus, or improper food in the stomach, a congested state of the liver, feculent accumulations or worms in the intestines, or disordered uterine function, become common causes of sympathetic irritation of the heart. To this class may be added all fevers, and those inflammations of any part of the body which are attended with increased action of the heart. In many of these, the quality and quantity of the blood may also contribute to the irritation, by a direct effect on the heart; and this organ may likewise sometimes acquire increased irritability, which conspires to quicken its action; but the earlier and more general cause of the excitement of the heart is, most probably, sympathetic. In all these instances the increased action of the heart is but a symptom; and although a very important one, as indicating the amount of the irritation, and tending to spread its effects throughout the system, it can scarcely be considered as even a functional disease of that organ, which it leaves healthy and uninjured, only perhaps weaker from the fatigue of long excitement, as it would have been from the excitement of exercise.

3. Although, however, the inordinate action of the heart induced by the two classes of causes to which I have adverted may be considered as only symptomatic, and secondary to conditions foreign to the heart, yet it puts to a trial the capacities of the organ, and if there be any defect in its power or in its structure, it is under the circumstance of unusual excitement that this is likely to be detected. Hence a heart that is unnaturally irritable, whether from functional or structural causes, is excited by any of these sources of irritation more readily and strongly than one in a natural condition; and it not unfrequently happens that it is only by their additional influence that the extraordinary irritability becomes manifest. This consideration has introduced to us the third class of cases of increased action of the heart—those in which its irritability is increased, and which are, more properly than those hitherto noticed, of the number of cardiac diseases. The irritability of the heart, as of other organs, may be exalted by two conditions which are developed in very different circumstances: one may be called nervous irritability, because it is associated with other phenomena of a nervous character, in nervous, hysteric, or epileptic subjects, and in those in whom a train of nervous symptoms have been developed in consequence of excessive blood-letting, or inanition of any kind, which tends to give an undue preponderance to the nervous function. The other condition of the heart which increases its irritability belongs more properly to the vascular function, being that which accompanies the various degrees of vascular distension or the inflammation of its tissues.

These several circumstances inducing increased action of the heart may occur separately, but they more commonly occur combined, or

they lead to one another. Thus the heart, when long irritated unduly, often acquires an increased irritability ; whilst at the same time being, like other muscles, fatigued by the excess of exertion, its contractions are weak although frequent. You may find an example of this in the effect of excessive or long continued exertion : a smallness and frequency of the pulse may continue for a long time, and contribute to the protracted weakness and prostration which great fatigue so often induces in delicate frames. Again, long continued irritation of the heart by plethora or congestion is very apt to induce a more permanent morbid condition of the organ ; thus the increased action arising from continued distension of the heart with blood is calculated to modify its irritability, and especially disposes the tissues to change. Such a plethoric or internally congestive state is a common forerunner of gout and acute rheumatism, and exists in other instances in which the excretions are defective or depraved. I have met with the signs of it in several cases where the secretions of the liver and the kidneys were at fault ; how far secondarily I cannot take upon myself to say, but the distended size of the heart as measured by percussion, and its increased action, sometimes breaking into irregularity or palpitation, continued until the internal plethora was reduced, temporarily by blood-letting, and permanently by a restoration of the due quantity and quality of the excretions. It is especially where the vascular functions are chiefly disordered, even independently of inflammation, that excitement of the heart is likely to lead to more permanent disease either of its properties or of its structure. I have known the heart to continue more or less excited, sometimes to an excessive degree, for many days in succession, nay for weeks, from merely nervous causes, without any permanent mischief ensuing. I remember an hysterical girl, whose pulse was from 120 to 160 for nearly three weeks : she complained of no other inconvenience than weakness, short breath, and a feeling of *flurry* ; and the pulse gradually subsided to a perfectly healthy standard of about 80. But when this excitement is more peculiarly that of the vessels, and even when it is of the mixed character which is presented by many kinds of prolonged febrile irritation, the heart continues to be irritable and weak for a considerable time, and if it do not show some permanent derangement of its properties or structure, its defective power becomes manifest through the period of tedious convalescence which generally ensues. I sometimes meet with patients who complain of the tardiness of their recovery from a fever or some other severe illness ; the pulse is quick, but weak, and easily accelerated ; the sound of the heart is loud, but brief, and its size, as determined by percussion, is large ; it has not the strength to contract fully on its contents, and unless these be diminished, or its strength be improved, some ulterior consequences of its imperfect function, and a consequently defective circulation, may ensue. Whether evacuants for the former purpose, or tonics for the latter, or both combined, shall be best suited to remedy the evil, will depend on other features of the case, and the condition of other organs, which

must all have a due share of attention ; but I now adduce the case as an example of another origin of cardiac disorder.

You may perceive that a comprehensive view of the functional pathology of the heart would embrace the numberless cases of disease in which its action is modified, which would form a large proportion of any nosological catalogue ; but this would be far beyond the purpose and limits of these lectures, and I have adverted to this bearing of the subject only to remind you of the very numerous relations of this most important organ, and the various modes in which these relations may become the channels of its idiopathic as well as of its sympathetic affections. The study of the causes of functional disorders of the heart is further important in relation to its structural lesions ; for the most distressing and dangerous effects of these often depend on the extent to which the action of the heart becomes disordered by additional functional disturbance, rather than by the amount of the permanent structural lesion. Hence slight organic disease in an irritable heart, or one readily disturbed by sympathetic relations, will be more severely felt than a more considerable lesion in one whose properties are less excitable. We shall have occasion to advert to this again, but we must now examine a little further the phenomena of functional disease.

The general symptoms of inordinate action of the heart are commonly either *palpitation*, which is the action of the heart perceptible to the patient, and is sometimes very distressing ; or a feeling of fluttering, agitation, or anxiety, within the chest, without any distinct throbbing. There is frequently greatly increased action, without the patient being conscious of any palpitation ; the feeling complained of is rather breathlessness, although the affection of the breath is only secondary. It is great violence, rather than frequency of the pulsations, that renders them perceptible to the patient : and if we take this character as constituting palpitation, we may, contrary to the statement of Laennec, have palpitation with a slow pulse. In fact, the feeling of palpitation may be induced by augmented sensibility of the heart or of the adjoining parts, when there is very little increase of action ; and many sensitive and nervous persons can always feel the motions of the heart independently of any inordinate action. The feeling of palpitation cannot, therefore, be taken as a general measure of the action of the heart, but rather of its sensibility. When the sensibility is great, there may be pain with the palpitation ; and this is sometimes very acute, being of the lancinating, constricting, or burning kind, shooting to the back, left shoulder, and arm ; which is called *angina*. Sometimes it is duller, and more like the feeling of soreness. The pulses of an over-active heart are sometimes felt in some of the chief arteries more than in the organ itself : this is especially the case in the carotid and vertebral arteries, in the arch and descent of the aorta ; hence the throbbing in the head, neck, at the top of the sternum, and in the epigastrium, which is sometimes as distressing as palpitation. We often observe that the chief force of inordinate action of the heart seems to be expended in the first

parts of the arterial tube, and does not reach the extreme branches, the pulses in which are not unfrequently weaker than usual. For the same reason also, whilst the head is hot and the face flushed, the extremities are often cold, or if they retain heat, they are dry and unperspiring. Hence, too, if the inordinate action be prolonged, may arise disorders of the secreting and nutrient functions: the irregular distribution of blood overloading the vessels of some parts, and imperfectly supplying those of others.

On physically examining an excited heart, we find the impulse and sounds much increased, as after violent exertion. We feel it abruptly and forcibly knocking against the ribs; but if it be not enlarged, we can perceive that its stroke is short and circumscribed, like that of a moderate sized body, and we find on percussion that the extent of dulness is as limited as usual. The first sound is shorter than usual, and so loud that it can often be heard without the actual contact of the ear or the stethoscope with the chest. The second sound is sometimes also loud; but if the pulsations be very rapid, which they sometimes are by double or treble their usual rate, the second sound becomes indistinct and a mere appendix to the first; the quantity of blood thrown into the arteries at each pulse being insufficient to make their valves flap freely and with force. When the action of the heart is increased in force more than in frequency, which occasionally happens in excitement from plethora and sthenic conditions of the vascular system, the impulse is strong, but less abrupt; the first sound longer, and the second sound louder, than where the pulsations are frequent. These characters are also permanently found in the pulsations of some enlarged hearts, but then there is more dulness than usual on percussion in the region of the organ. In violent palpitation, the enlarged pulsations of the arch of the aorta may be felt and heard loudly at the top of the sternum, those of the subclavians below the clavicles, and of the carotids in the neck, where also the jugular veins are often seen pulsating, from the retropulsive action of the right ventricle.

When the inordinate action of the heart is excited by transient causes, its duration may be temporary, constituting what are called fits of palpitation; and the heart, when permanently diseased, is frequently worked into these fits by slight additional irritations. They are often most painful and distressing; being attended with feelings of oppression, of extreme faintness, of indescribable agitation, &c.; sometimes of severe anginal pain, cutting or tightening across the chest, and sometimes shooting to the back, neck, and arms. They are in some cases aggravated by fits of spasmodic asthma, of which they may be either the cause or the effect. These formidable symptoms belong chiefly, but not exclusively, to organic diseases of the heart.

When inordinate action of the heart depends on inflammation increasing the irritability of its tissues, it is more permanent in its duration, partaking of the course of the inflammation. But as the inflammation does not affect all parts of the heart alike, some may

become more irritable than others, and contracting more abruptly, or with greater force, may modify the character of the systole and its sound. This happens especially when the irregular irritability includes the muscular pillars which regulate the motions of the mitral valve. If these act too suddenly, but perfectly, the flapping commencement of the first sound may only be exaggerated ; but if they act irregularly, so as not, with the pressure of the blood, to close the orifice of the auricle, the blood will regurgitate through it, causing a blowing murmur, which is generally heard best where the apex is felt beating. This regurgitation, if considerable, may develop another cause of excitement in the obstruction which it causes to the onward progress of the blood : this subject we shall notice under the head of valvular disease.

I think we might well class together the cases of *irregular* action and those of *defective* action of the heart ; for in almost all cases of irregularity, if the action be not defective, the effective power is. The irregularity may be either in the *rhythm* or *period* of the pulsations, or in their *strength*, or in *both*. These present great varieties, but I do not consider it profitable to occupy your time by too many distinctions, and I would therefore only notice those that may illustrate the properties of the heart, or suggest some practical inference.

Slight irregularity in the rhythm of the heart is by no means uncommon in persons who are weak or nervous. Thus we often find a pulse now and then occur sooner or later than in the natural order, constituting *reduplications* or *intermissions* of the pulse. Such phenomena are very common in those who are suffering from much bodily fatigue. I have also known such an irregularity produced at will, by assuming a constrained posture or by holding the breath. They may occur quite independently of disease of the heart, although they are also frequent concomitants of it. An intermittent pulse may be said to be constitutional in some persons ; but they generally have other signs of a weak or an easily disordered circulation. When there is no disease of the heart, the sounds of the irregular pulsations are natural, except that those which occur before their time may be shorter and more abrupt, and the retarded ones are stronger and more prolonged than usual, which are the simple results of the different quantities of blood which the contractions have to propel. In the absence of signs of other disease which may cause it, simple irregularity of the pulse may be considered a result of defective tone of the heart and vessels, and may sometimes be taken as an indication for the use of animal food and tonics.

Irregularity in the strength of the heart's pulsations commonly depends on organic disease. It may be more evident in the arterial pulse than it is in the region of the heart, for being there reduced by distance, the weaker beats may be scarcely, or not at all perceptible ; and the artery, consequently, often has a pulse much less frequent and regular than the heart. Many of the instances that have been recorded of extremely slow pulse are probably of this kind. I have met with several cases in which the pulse at the wrist was below 30,

and irregular, when that of the heart was 60 or 90, and pretty regular as to rhythm, but varying much in strength, so that the strong beats only reached the wrist. But it is more common for the rhythm to be also disordered, so that on listening to the heart, instead of beats at regular intervals, they occur in all varieties of measures, very unequal both in time and strength; and at the wrist the inequality is perhaps still more evident. Amidst all this irregularity, when perhaps no three consecutive pulses are of the same period or strength, there is very often something like an attempt at order, every third, fourth, or fifth beat being stronger, and followed by a longer interval, so that a sort of tattoo measure is more or less kept up. In persons whose pulse only occasionally presents these irregularities, we may sometimes observe another curious phenomenon: the heart which was beating regularly at 60, will suddenly double or treble its number of pulsations, still preserving its regularity at 120 or 180, producing the feelings of flurry, oppression, faintness, or angina, that have already been noticed as attendant on inordinate action. After a while the pulsations may, as suddenly as they quickened, fall back to their slow regular standard. But sometimes, instead of this sudden and complete change of measure, the two rates of pulsation seem to be variously mixed; three or four beats of the quick movement being followed by a strong one of the slow, and a pause, or some other such combination. This is a common kind of the permanently irregular pulse that we meet with in certain forms of organic disease of the heart, particularly those affecting the left auricular valve; and in the tendency to periodicity in the recurrence of the stronger and slower beats, we still see the remains of a natural rhythm, which prevails most in the more tranquil conditions of the circulation. I have known cases, however, in which a pulse habitually irregular became regular during the prevalence of feverish irritation; but the converse is more commonly observed, that the irregularity is increased with excitement. Thus it often happens that pericarditis, supervening on organic diseases of the heart, increases the irregularity, or occasions it where none existed before. In these more transient forms of irregularity there is not the measure or period in the unequal beats which is manifest where the irregularity is more habitual; it would seem more of the varying character of the excitement of a heart that has lost its natural rhythm without having had time to form a new one; whereas in permanent irregularity the heart has, under its circumstances of varying excitement, become endowed with a new and more rapid rhythm, between which and its natural one it is continually fluctuating, in something like a regular order. This is an exemplification of that tendency which the animal body so much exhibits, to retain some order even in the midst of anomalies.

On listening to the pulsations of a heart acting irregularly, you may find a good illustration of the principle that the character of the sounds depends in great measure on the mode of action. Here is

the same heart giving in succession a considerable variety of sounds, the quick pulses being short, loud, with little impulse, and without a second sound; and the slower ones every now and then coming in with a longer heavier sound, strong impulse, and with a second sound, whilst the valvular disease that is frequently also present in these cases may add a murmur which modifies some or other of these sounds, according as it may occasion the blood to pass in an unnatural manner. Commonly the short quick pulses, belonging to the high rhythm prevail the most, the strong pulsations being only occasional; but sometimes in a pulse which is slow and pretty regular, there will be now and then two or more minor quick pulsations, which often do not reach the pulse at the wrist. These being short, and unaccompanied by a second sound, were supposed by Laennec to be repetitions of the second sound, which he referred to the contraction of the auricles. But they may often be felt in the carotids, and are thus proved to be minor ventricular pulses; in fact, they exactly resemble, and are examples of, the short rapid pulsations of a high rhythm. When this rhythm prevails altogether, to the amount of 169 or 200 in a minute, there is such a vibratory commotion in the region of the heart, that one is almost tempted to suppose that the two ventricles are acting alternately; but this supposition is hardly tenable when we consider their fibres are in great measure continuous, and not adapted for separate action. In judging of the size or strength of a heart acting irregularly, we must attend chiefly to the strong slow pulsations, which best represent the true capacity and properties of the organ; for the frequent rhythm, with short sound and no impulse, may take place in any kind of heart, large or small, being perhaps produced by a twitching or quivering movement of the fibres, rather than a real and complete systolic action. The strong pulsations also may inform us by the murmur which may accompany them, as to the condition of the orifices. Lesions of some part of the valvular apparatus of the heart are generally the foundation of permanently irregular action, although its immediate recurrence may be determined by more transitory causes. Of many cases of habitual irregularity in old people I have never failed to find signs of disease of the orifices or valves to a greater or less extent, although in several instances the constitution had, in a measure, adapted itself to the anomaly, and the individuals suffered only occasionally from shortness of breath, palpitation or fluttering at the heart, faintness, or some similar symptoms of inefficient central circulation. But these persons cannot undertake great exertion without suffering much from it; and attacks of catarrh, or such affections, which might be trivial in others, fall heavily on them. I have met with several examples of irregular action of the heart beginning after unusual bodily exertion and mental excitement, chiefly in stout persons, and in those of a gouty habit. In most of these the irregularity subsided in time, after quiet moderate living, and a due action on the secretions; or more rapidly on the appearance of gout in an extremity. In one case the irregularity has become habitual, without, however, material disorder of the general health, although

there is evidently a slightly defective action of the left auricular valves. We shall have to notice such cases by and by.

I shall detain you with but few remarks on the subject of *defective* action of the heart, for unless it be accompanied with irregularity, or amount to syncope, it is more commonly a part of general weakness of the whole system than an affection of the heart in particular.

The action of the heart may fail in force or in frequency, or in both ; and its failure in either respect, when extreme, produces the phenomena of *syncope* or *fainting*. Some circumstances render the action of the heart very slow, intermittent, and may stop it altogether ; such are the influence of some sedative poisons, such as digitalis, tobacco, and some fungi ; sudden mental emotions ; severe injuries to the nervous system, as by the removal of blood from the brain by change of posture when the vessels are relaxed or ill filled ; or by a strong shock to any considerable portion of the body, as by crushing a limb, by a blow upon the epigastrium, or by the ingestion of a large bulk of cold liquid, or of various articles which are noxious from idiosyncrasy. These and similar causes appear to act by diminishing the irritability of the heart, so that its contents do not stimulate it as usual. Other causes impair the heart's action rather by withdrawing its proper stimulus, and by diminishing the strength of the contractions which become very frequent before they fail ; this is the case in syncope from hemorrhage, and from various inflammatory and febrile diseases, especially those of the abdomen, which commonly prove fatal in this way. Syncope of either kind may be induced in cases of diseased heart, and be the mode of their fatal terminations, either quite suddenly, or by permitting the formation of a polypus or fibrinous coagulum, the obstruction of which may cause death in a few hours. A great variety of other causes, mental and physical, may produce syncope, in those that are predisposed to it.

The general symptoms of syncope are, feelings of sickness, giddiness, swimming in the head, noise in the ears, indistinct vision, sudden pallidity or change of the countenance, occasionally with quivering of the lip, chattering of the teeth, involuntary groaning, slight convulsion, and ultimately loss of consciousness and apparent death, which may last for some seconds, minutes, or even hours. Restoration is often accompanied by the breaking out of perspiration, eructation of wind, vomiting, slight convulsions, or palpitation ; and sensations far more unpleasant than those which preceded the loss of consciousness, and which some have described as rather agreeable than otherwise. The radial pulse is either weak or altogether imperceptible during syncope. The sounds of the heart are also commonly feeble, very short, and generally without a second sound : sometimes they are frequent, in other cases very slow, and they are generally irregular or unsteady. I have never met with a case of syncope in which they were inaudible ; but we can readily conceive that pulsations may take place with so small a force as to produce no audible sound, and yet to propel the blood enough to preserve life. In some of my experiments on animals, slight contractions recurred for some time

after all audible sound had ceased ; and in one instance the auricles alone continued to act alternately for several minutes after the ventricles had lost all motion. It is possible that by either of these remnants of power in the central organ of circulation, with the aid of the powers which assist in the capillary vessels, there may be kept up a movement of the blood sufficient to sustain such a low degree of organic life as may resist decomposition, and keep the frame for a time in a condition from which it can be again excited to its proper standard of vitality. This condition of prolonged syncope, or suspended animation, closely resembles the torpid condition of hibernating animals, which I had occasion to notice in a former part of this course.

But the action of the heart may be defective, yet short of the degree which causes the phenomena of syncope. This defective action may be caused by the same circumstances which occasion syncope, in which case it may be of temporary duration ; but it may also arise from a more permanent weakness or defective contractile power of the heart. Such a weakness may be the effect of low diet ; of long-continued illness, especially those attended by much vascular excitement, such as fevers ; of long breathing vitiated air ; of sedative medicines, such as digitalis, hydrocyanic acid, &c., and, lastly, the heart may be constitutionally weak, from natural deficiency of its size or strength, in proportion to the body through which it has to propel the blood. Under any of these circumstances there will arise more or less of the general effects of imperfect or languid circulation of the blood. Such are, coldness of the extremities, often attended with flushing or a feeling of heat in the head and face ; a general feeling of languor, with pains in the back and limbs ; shortness of breath, and fainting on slight exertion ; œdema of the feet, sick headaches, a loaded tongue, weak digestion, constipated bowels, and a defective or disordered condition of all the secretions. The pulse is weak and small, and so is the impulse in the region of the heart, although it may be abrupt ; and the accompanying sound is pretty loud, but short. The action of the heart may be either frequent or slow ; but it is most readily excited by exertion or any other cause to palpitation or other kinds of irregular inordinate action. It is often this palpitation that alone draws attention to the heart ; and not unfrequently a lowering treatment has been adopted to reduce this inordinate action, when it has all the while been the result of the mere weakness of the organ, which struggles at a task to which it is unequal. An excitable condition of the nervous system sometimes accompanies this weak state of the circulation, and may variously modify it, increasing the irritability of the heart, but not its power. Such a combination is often met with in chlorotic females, in whom also the defective quality and quantity of the blood form another conspicuous morbid feature. Weakness of the heart not uncommonly manifests its effects in those who have grown rapidly, whether in height or bulk. Young persons who have shot up in stature in a short time without a proportionate increase of strength, sometimes suffer from palpitations and shortness

of breath on exertion, and their generally weak pulse, cold hands and feet, with liability to chilblains, and livid redness of the nose, are effects of languid circulation. So, also, individuals of a very different description sometimes suffer from the same cause; those who have become very fat. The short breath and palpitation which such persons often suffer from, may partly arise from the deposition of fat in the mediastinum and around the heart, encumbering its movements; but in part they also depend on the inadequacy of the heart to the increased weight of matter through which it has to propel the blood. Hence such subjects seldom bear well bleeding, or any other kind of sudden lowering influence, which does not at the same time reduce the bulk of the body. After a while, under favourable circumstances, the heart acquires such an increased size and strength as to enable it to distribute blood more effectually throughout the body, and in proportion as this takes place, the individuals, although as stout as ever, gain more activity and strength of circulation, and no longer suffer in the same degree from palpitation or shortness of breath. And should they suffer a sudden diminution of their bulk, the heart will then be too strong for their bodies, and they will suffer from the symptoms of hypertrophy or enlargement of the organ which we shall have occasion to notice hereafter.

The general symptoms of many organic diseases of the heart are also those arising from deficient effective action of the organ, although its apparent power may at the same time be very great. It is of great importance to hold this in view in relation to the treatment; for in many cases of such affections, as well as in others in which inordinate action arises from want of power rather than from its excess, more benefit may be derived from a judiciously directed tonic plan, with especial precautions not to overtax the weak organ, than from measures directed against its occasional excitement.

We have not time to give a distinct notice of the exalted sensibility of the nerves of the heart, which causes the phenomena described under the names, *angina pectoris*, *syncope anginosa*, *sternalgia*, &c. Pain in the region of the heart, accompanied by a feeling of faintness, is in truth rather a symptom than a disease; and it may occur in various degrees in various affections of the heart and great vessels, functional as well as organic. The kind of pain, cutting, piercing, tearing, burning, crushing, shooting to the back, arm, and throat, and so forth, as it is described in different cases; the suddenness of its attacks, and the temperament of the individuals in whom it occurs independently of organic disease, evidently refer it to the class of neuralgic pains. I have known it in a very severe form attack a patient who had before been a sufferer from *tic-douloureux*. It is commonly associated with organic lesions, especially, but not exclusively, that affecting the aorta and its valves; but such affections frequently exist without it. So, on the other hand, anginal or neuralgic pains sometimes occur in persons who have no organic disease; thus resembling other forms of nervous disorder; which, although occasionally excited by the irritation of bony deposits, or other per-

manent lesions, may arise from an excessive sensibility developed by more transient causes. The circumstances which generally determine the immediate attack of a paroxysm of angina are those which excite the heart's action, such as walking quickly up a stair, up a hill, or against a strong wind, strong mental emotions, distension of the stomach, and the like. To avoid these circumstances, and to employ measures which may lower the nervous sensibility of the heart, form the chief indications of treatment ; indications which will be the more difficult to fulfil, in proportion as the organs are affected by a permanent disease. We have so little time left for the consideration of diseases of the heart, that I think it best to condense into one final lecture what I have to say on their treatment, which will prevent repetitions, and enable me to give more in one comprehensive view.

LECTURE XXVII.

Diseases of the Heart (continued.)—Inflammation of the Pericardium.—Anatomical Characters of Pericarditis.—General Symptoms ; their Uncertainty.—Connexion of Inflammations of the Heart with Rheumatism.—Physical Signs of Pericarditis.—Friction Sounds.—Signs of Effusion, &c.—Varieties of the Disease.—Adhesions of the Pericardium ; their different Kinds and Effects ; their Signs.—Inflammation in them.—Inflammation of the Endocardium.—Anatomical Characters of Endocarditis.—General Symptoms.—Pathology and Physical Signs.—Frequent Occurrence of Endocarditis.—Its Constitutional Effects.—Inflammation of the Substance of the Heart.—Symptoms and Signs.

INFLAMMATION of the heart produces different symptoms and consequences, according to the structure which it chiefly affects. It commonly attacks the serous and fibrous membranes either covering the exterior of the organ, or lining its interior, and by their folds forming the valves ; more rarely it affects the muscular fibres, or the cellular texture which unites them. Hence the division of inflammation of the heart into the species, *pericarditis*, *endocarditis*, and *carditis*. The two first are commonly combined, but not constantly ; and we may well consider them separately.

Inflammation of the Pericardium, as of the Pleura, tends to produce an effusion of serum and coagulable lymph on the free surfaces of the membrane ; by these products, as well as by other changes of the vital and physical properties of the heart, pericarditis develops various symptoms and signs, by which we may ascertain its existence.

I do not consider it necessary to describe to you at length the various morbid appearances which are found after death in the inflamed pericardium. They vary like those of pleurisy, and I refer you to the descriptions which I have given of them. There is this difference, that the motion of the heart being constant, and not like that of the lung interrupted by the effusion, it in various ways modifies the de-

position of lymph, according to the quantity of accompanying liquid ; and thus are produced those transverse ridges and furrows, honey-comb or reticulated, and shaggy or villous appearances, which are seen in the lymph of an inflamed pericardium, and which Laennec imagined to be a peculiar kind of new structure. The liquid may be serous, sero-purulent, or purulent ; with all the varieties that are found in the liquid of pleurisy.

The general symptoms are often very obscure. There may be pain below the left breast, shooting to the sternum, back, and left arm ; perhaps with tenderness on pressure on or between the cartilages of the fourth, fifth, and sixth ribs, or on the epigastrium ; but all such pain or tenderness is sometimes entirely absent, and is more commonly produced by pleurisy than by pericarditis. Neither is dyspnœa nor palpitation always present, although they are sometimes extremely distressing, and give the patient's countenance an expression of anxiety, uneasiness, or restlessness, which is very characteristic, and may be perceived even when the patient does not acknowledge the existence of the sensations. I have often known pericarditis to exist without producing in the chest any unusual sensation whatever ; and even the pulse in the first stage has been below its usual frequency. Besides this, these symptoms are occasionally disguised by delirium, or a state of incoherent stupor, which draws all the attention to the brain, although it is only secondarily affected. Under these circumstances it is not wonderful that the existence of the disease should have been often overlooked, and known only through an examination after death, or by the production of a permanent organic disease, which is more unequivocal in its symptoms.

But pericarditis cannot exist for any length of time, and certainly not to a dangerous extent, without producing physical signs that are quite characteristic. That these have often escaped notice, even of auscultators, is because they have not been sought for. It is of great importance, therefore, to examine the region of the heart in all cases in which there is the probability of its being affected, whether any symptoms have manifested themselves or not. The cases in which you should especially be on the look out for the signs of pericarditis are those of rheumatism in all its forms, but particularly the acute kind, which is generally called rheumatic fever ; the subjects of mechanical injuries, or of pleurisy or pneumonia affecting the left side ; and all doubtful instances of difficult breathing, especially when accompanied by œdema, general or partial. Of the causes of pericarditis, rheumatism is by far the most common ; but it still more frequently produces endocarditis. In fact, although I do not agree with Bouillaud's opinion that these inflammations are essentially a part of rheumatism, I can confidently state that I have found signs of endocarditis or pericarditis, or both, to a greater or less extent, in fully three-fourths of the cases of rheumatism which I have examined in the last three years, although in little more than half that proportion was there any complaint of pain in the chest, palpitation, or dyspnœa. That the membranes of the heart are frequently affected in

cases of rheumatism has been long acknowledged by English practitioners. In the first volume of the *Medico-Chirurgical Transactions*, Sir David Dundas published a paper expressly pointing out this fact ; and Dr. Wells has given many illustrations in the third volume of the *Transactions of a Society for the improvement of Medical and Surgical Knowledge*, published in 1812. Yet M. Bouillaud, not familiar with English medical literature, or having consulted only recent works, which were imperfect in their information, has imagined himself to be the first to point out the connexion between rheumatism and diseases of the heart. We are, however, indebted to him for much valuable knowledge with regard to diseases of the heart in general, and for better information than we possessed before the publication of his work, as to the kind and extent of share which rheumatism has in producing diseases of the heart. I must refer you to his work, which is the most complete on the subject, although it is vitiated by a spirit of hasty and excessive generalization, which too much prevails in the Broussaian school of medicine. I have great pleasure in referring you also, for the most complete digest of modern information on diseases of the heart, to the fourth part of Dr. Copland's admirable *Dictionary*, which has just appeared (May 1837). Each subject that this enterprising writer undertakes he masters so completely, as to outdo even those who have made it the chief object of their study. The observations which I shall have to offer you are chiefly the results of my own experience and study ; for a complete or historical view of the subject I refer you to Dr. Copland.

The first physical sign of inflammation of the membranes of the heart is an increased loudness of the natural sounds, with an impulse stronger and more abrupt than usual. The pulsations are generally but not always accelerated ; sometimes they are slower than usual, and then they are stronger ; and the first sound, although loud, is prolonged even when there is no murmur. Sometimes there is now and then a reduplicated pulse, or some other slight irregularity ; but the beats are for the most part regular, and they owe their loudness to their increased vigour. In this state of the signs the inflammation may be in the pericardium, endocardium, or both, without exhibiting any thing distinctive of its seat for from twenty-four to forty-eight hours, and may sometimes be removed by general and local blood-letting, without proceeding further. But in that time, and perhaps in less, new signs commence, which announce changes which distinguish the seat of the inflammation. Those of endocarditis are various murmurs, which are commonly the first to show themselves ; but we must consider them afterwards. Those of pericarditis are various sounds of superficial friction, which are quite characteristic. At first this sound is soft and rustling, like the rubbing together of two pieces of paper or silk stuff ; and it may accompany only part of the natural sounds, from which, however it is obviously distinct, in being much more superficial. It is generally heard first about the middle of the sternum, or to the left of it, corresponding with the base of the heart, or the attachment of the auricles. It afterwards increases in

loudness and duration, being heard beyond the immediate region of the heart, and accompanying not only the periods of the natural sounds, which it disguises, but also the interval between them. It thus gets a sort of continuous jogging rhythm, corresponding with the movements of the heart, which is like that of the saddle when one rides on horseback; and when, as it generally happens, the friction sound becomes harder, and more like the creaking of leather, its resemblance to the noise of a new saddle is quite ridiculous. In some cases the noise is crackling, like that of crumpling dried membrane or parchment. We owe the discovery of this valuable diagnostic sign to M. Collin; and it has more recently been described by Reynaud, Drs. Stokes and Watson, and others. I have had many opportunities of observing it in its different modifications, and of verifying its accuracy by examination after death, during the last five or six years. I only wonder why Laennec and Bouillaud did not recognise it, even after M. Collin had described it in 1824. They must have mistaken it for the double grating murmur of diseased aortic valves, from which, however, its more superficial, diffused, and less regular character, would easily distinguish it. The confirmed leather creak sound is certainly caused by the rubbing of the rough coating of lymph on the pericardium proper, and on its sac; but I have some doubts whether effusion of lymph is necessary to produce the rustling sound, which occurs earlier, and whether it may not be caused by a mere turgescence of the vessels, and deficiency of the natural serous lubrication, which might be supposed to take place in the early stage of inflammation.* The natural sounds of the heart are completely disguised by the friction sound; but they may sometimes be heard at the top of the sternum, and in the carotid arteries, and they will be often attended with a murmur; this depends on the simultaneous existence of endocarditis, and will be noticed presently.

The pericarditic friction sound does not commonly continue for many days in succession. Either the lymph is absorbed, or it is converted into false membrane, which forms a bond of adhesion more or less perfect between the heart and the sac, in which case the sound gradually ceases; or if the inflammation continue in a low degree, serum is poured out in such abundance, that the heart moves free in the distended sac, and no longer rubs against it. In either case the friction sound becomes less constant, accompanying only the stronger pulses, and gradually ceases. In case of adhesions it is commonly heard longest below the left breast; and when it is terminated by liquid effusion, it generally continues latest near the sternum,

* Some experiments have been recently performed for the British Association, by Drs. Clendinning, Todd, and myself, with a view to determine the physical cause of the friction sound. They have led us to the conclusion that inflammatory injection of the pericardium is insufficient to produce it: but the smallest effusion of lymph, and even slight ecchymoses, or effusions of blood under the pericardium proper, from acupunctures, may cause distinct rustling or rubbing sounds. A very good example of the leather sound was presented by a heart which was found to be covered with a thin film of quite soft lymph.

corresponding with the base of the heart, which is less easily separated from the sac than the apex. In the latter case change of posture, as by leaning forward, will also sometimes reproduce it when it has ceased. I have known it in a few cases to continue for more than a fortnight in conjunction with the signs of liquid effusion; for this effusion does not always proceed to an extent sufficient to separate the heart from its sac, particularly at its anterior surface.

You now know enough of the principles of acoustic signs to perceive that the effusion of serum in the pericardium must produce dulness on percussion in the region of the heart; whilst, if copious, by removing the heart from the walls of the chest, it generally renders the sounds more distant, and more or less impairs the impulse. The dulness may be limited to an area of two or three inches at the lower part and to the left of the sternum; or in case of extensive effusion it may occupy the whole front of the left side of the chest, even as high as the second rib, and extend even to the right of the sternum. There being no sound of respiration, and sometimes a prominence of the ribs in these parts, the case might be taken for pleuritic effusion, but that the sounds of respiration and percussion are still good in the back and in the axilla, and are not materially varied by the change of posture. There does not seem to be any fixed rule which regulates the displacement of the heart by liquid in the pericardium; for although, as I have just told you, this organ is commonly displaced in proportion to the quantity of the effusion, so that the friction sound is stopped, and the impulse and natural sounds are rendered distant, yet I have met with cases in which extensive dulness on percussion, indicated copious effusion, yet the friction sound and impulse still continued strong at the sternum. We may discover when the sounds are rendered weak merely by displacement, on listening to them at the top of the sternum and in the carotid or subclavian arteries, where they may be heard of their usual intensity. This enables us to distinguish this case from that of a greatly enlarged heart acting very feebly: in this instance there would be extensive dulness on percussion with weak sounds and impulse, but the sounds would also be weak in the course of the arteries. When the liquid in the pericardium is scanty, it may only partially intercept the impulse, and modify rather than obscure the sounds. Thus in the standing or sitting posture the impulse at the apex is diminished, and the sounds assume that more drum-like character that I have before described as the effect of an intervening layer of liquid; whilst at the sternum both sounds and impulse may be as usual. In the supine posture similar differences may be presented in an altered relation; the drum-like sounds and diminished impulse corresponding with the dulness on percussion.

With all the signs of extensive disease that I have been describing, the general symptoms may be combined in most varying and uncertain proportions; being sometimes well marked, and necessarily drawing the attention of the practitioner to the seat of the disease; in other cases being so trivial or equivocal as scarcely to be acknow-

ledged, even by the patient. Some feeling of dyspnœa or faintness, especially on moving, is the most constant symptom ; and this is generally accompanied by irregularity of the pulse. The contractility of the heart, which was in the first instance exalted by the inflammation of its membranes, ultimately becomes impaired ; the action loses its rhythm, becomes sometimes palpitating, sometimes defective, and verges on syncope ; syncope, in fact, sometimes does occur, and may suddenly terminate the patient's life. But this is by no means a common case. I have met with but few cases of pericarditis which first attacks a sound heart, proving fatal in its acute or chronic form ; but I have known several partially or apparently recover from a first attack, after some months or even years exhibit signs of organic disease of the heart, and ultimately sink under a subsequent attack of pericarditis supervening on this. A large proportion of cases, even where the disease was very extensive, recovered entirely, as far as their subsequent history was ascertained. I do not, therefore, look on pericarditis as so formidable a disease as it has been generally considered to be. In fact, its existence has hitherto been known chiefly by those instances in which it has either proved immediately fatal, or has become conjoined with serious organic disease, which constitute but a small part of the cases which we can detect by physical diagnosis.

The course of pericarditis is, like that of pleurisy, very much determined by the nature of the effusion. When this is moderate in quantity, and composed equally of serum and healthy lymph, it may all be absorbed, and the cure completed in a short time. Such we may conclude to be the case when the friction sound and dulness on percussion last only two or three days, and no sign of disease of the heart remains. When the lymph is more abundant, adhesions will form between the heart and pericardium ; and the character and effects of these adhesions will vary mainly according to the quantity of the liquid effusion, and the manner in which this protracts the period of their formation. If the liquid be moderate in quantity, or soon diminish so as to bring the lymph early into adhering contact before its plastic qualities are impaired, and whilst the heart is still vigorous and free in its action, the adhesions formed will then be loose and mobile, composed of yielding cellular membrane, which may very little, or not at all, interfere with the motions of the organ. Such adhesions have often been found after death in cases where their existence had not been suspected during life, not having in any notable degree impeded the heart's action. But if the liquid effusion be abundant, and persist long, there is great fear that the lymph effused will not be formed into adhering contact until its vitality is impaired, when it is capable of forming only denser and more fibrous adhesions disposed to contract further, and when, the action of the heart being feeble and irregular, it cannot adapt them to its healthier and freer motions ; in such cases the adhesions formed, whether partial or universal, will for ever shackle the motions of the heart, and constitute a serious form of organic disease. Such adhesions are

most injurious when they confine the apex, naturally the most moving part: they do less injury, when they attack the base only; but in this case they are often combined with internal organic disease, arising from endocarditis. Not only the quantity of the liquid effusion, and the consequent slow formation of the adhesions, may conduce to their injurious character and effects, but probably in some measure the nature of the inflammation also. I think that I have seen worse results from pericarditis occurring in subjects affected with fascial rheumatism, than from that supervening in those with the more acute disease confined to the joints. Weakly and aged persons are likewise more likely to suffer from the longer duration of the inflammation in them, and the less assimilable character of its products.

From what I have just said, you may infer that *adhesions of the pericardium* according to their character may or may not produce signs by which they may be distinguished. When they are mobile, and composed of yielding cellular texture, they do not alter the positions, motions, or sounds of the heart, and therefore yield no signs. When, in consequence of their closeness and rigidity, they do interfere with the heart's motions, and thereby irritate it into inordinate action, it may still be difficult to distinguish this inordinate action from that from other causes, so long as the heart is nearly covered by the lung. I have sought in vain for the "jogging or tumbling" motion which has been stated to be characteristic of an adherent heart. Such motion results from irregular action, often exists without any adhesions, and in some measure depends on the motions of the lung differently affecting successive irregular pulsations. But it often happens in case of close adhesions, that prior to their formation the pericardial sac has adhered in its distended state to the walls of the chest at the left of the sternum, so that when the heart also adheres to the sac, it constantly pulsates in close contact with these walls. This combination of circumstances gives us very appreciable signs. In the first place the motions of the heart may be seen and felt much more plainly and widely than usual, drawing in the intercostal spaces at each systole. Then these motions, instead of being as usual intercepted by the expansion of the lung in a full inspiration, are always close to the walls of the chest; for these walls, instead of, as usual, rising from the heart upwards and outwards at each inspiration, carry the heart with them in all their movements. Under these circumstances, therefore, there will be, proportioned to the adhesion and the size of the heart, a space in which the pulsations are always felt, and the sound on percussion is always dull in every stage of respiration and in every posture of the body. Compare this with what I told you of the shifting, varying force and position of the pulsations and the sound of percussion in the region of the heart in health, and you will understand the difference; and the distinction extends also in part to other diseases, which enlarge the heart, but do not fix it to the walls. When the heart is thus generally adherent to the pericardium, and this to the diaphragm and walls of the chest, the

enlargement of the organ which very commonly ensues cannot readily take place as usual, downwards and to the left, but it proceeds upwards and outwards, carrying with it the walls of the chest, to which it gives a remarkable projection about the ends and cartilages of the middle ribs. I have seen this accompanied by a retraction or hollow at the epigastrium. In other cases again, from the adhesions being more partial, the enlargement may take place laterally, and bring the pulsating apex of the heart far to the left side. Various other changes of position may arise from different circumstances, especially when the pericarditis has been conjoined with pleurisy.

It is a common notion among pathologists, that an adherent pericardium is in some degree secure from attacks of inflammation; but this is a great mistake. I have frequently found in both layers of the pericardium, and in the false membrane which unite them, traces of inflammation, such as punctuated and striated redness, softening of the membrane, and an effusion of lymph and a little serum, or a sero-purulent liquid, into their interstices, and into the adjoining cellular membrane in the mediastinum. Of course there can be no quantity of liquid effusion into an adherent pericardium; but under these circumstances this very commonly takes place into one of the pleural sacs instead. The exacerbations to which those affected with old rheumatic disease of the heart are occasionally subject, often originate in inflammation of the pericardium, or the internal lining membrane. An adherent pericardium has no immunity from these attacks; in fact, from its being so constantly fretted and strained by the inordinate motions of the heart, it may be conceived to be particularly liable to them; and if they occasion death, the appearances found are such as I have described. You may perceive that these inflammations of an adherent pericardium cannot give the signs that are usually distinctive—the friction sound, or the dulness and deficient impulse of effusion. Hence, unless their presence is indicated by local pain, tenderness, or soreness, it can only be suspected, on the occurrence of more than usual irregularity and excitement in the action of the heart, unaccompanied by other sufficient cause.

Inflammation of the internal membrane of the heart—*endocarditis*, as Bouillaud has very well named it, is a frequent but not a constant concomitant of pericarditis. Nor is it wonderful that inflammation should readily spread from one surface of the heart to the other, when we recollect how near they approach at the auriculo-ventricular and arterial orifices, where they are separated only by a fibrous structure of more strength than thickness. Now this very fibrous structure is especially obnoxious to rheumatic inflammation, and it is chiefly by inflammation of this kind that both surfaces are simultaneously attacked. Pericarditis, from other causes, may exist without any inflammation of the lining membrane; and endocarditis, when it exists independently of rheumatism, may continue long without affecting the pericardium; the inflammations in these cases being chiefly confined to the serous membranes, and neither affecting nor traversing the fibrous tissue between them.

The endocardium is placed in very peculiar circumstances with regard to the effects of inflammation upon it. It is a serous membrane, and the products of its inflammation might be expected to be like those of the pleura, or any other serous membrane, lymph and serum. But as we found that the continual motions of the heart give a peculiar form and character to such products in the pericardium, so these motions and those of the blood, must be continually acting on those of the endocardium ; in fact, they must be continually swept away ; the serum entirely, and the lymph also, except in circumstances very favourable for its concretion and adhesion to the secreting surface. It is no wonder, then, that endocarditis should not have been readily recognized ; since, in many instances, the traces by which inflammation is known to have existed could not remain to indicate its seat. So you may be prepared to expect that the anatomical characters of endocarditis are far less obvious than those of most other inflammations. We do, however, not unfrequently find in subjects dead of rheumatic and other inflammations of the heart, or of older organic disease, indubitable traces of inflammation of the lining membrane. Thus, especially at the left auriculo-ventricular ring, and in the membrane lining the auricle near it, as well covering the laminæ of the mitral valve as in the aortic valves, especially at their attachments and *corpora arantii* in their margins, we meet with striated or punctuated patches of vascular redness, sometimes accompanied by an inequality, roughness, or softness of the membrane ; occasionally, with soft thickening or distinct films of soft lymph upon it. In some cases we find apparent abrasions or distinct ulcerations of the membrane ; and to the edges of these, or to any roughness or irregularity which the valves or lining membrane may present, are attached little soft fibrinous bodies, generally of a ragged conical shape, and more or less tinged with blood, which have obtained the name of *vegetations*. These all are probably effects of the more acute form of inflammation. But the same parts often present changes which, in other structures, we are accustomed to refer to chronic inflammation, such as tough thickening, with partial puckering, induration, and even ossification, of the lining membrane, or of those folds of it which form the valves, and especially of the fibrous tissue that is a strengthening network in them. The tissues thus changed are liable to rupture, irregular distensions, and other accidents which we shall have to notice under the head of Diseases of the Valves.

The general symptoms of endocarditis are still more obscure than those of inflammation of the pericardium. There may be fever with increased action of the heart, sometimes with irregularity, more or less of the feelings of palpitation or faintness, oppression or agitation, and pain, heat or soreness, referred to the sternum or epigastrium ; but most of these may be absent, and associated as they often are with general rheumatic fever and pains, and occasionally disguised by sensorial disorder, or gastric and hepatic derangement, the secondary effects of the deranged action of the heart, the symptoms which should

fix attention on this organ in particular, are very frequently overlooked, or referred to the convenient and satisfactory category of *general irritation*.

If we examine the pathological effects of inflammation of the lining of the heart on the tissues of the organ, we see that it must do more than produce general irritation. If it be equally diffused over every part of the interior of the heart, it might, in the first stage, cause a general increase of irritability and action; but we have seen that it commonly prevails most in particular parts, especially at the aortic and mitral orifices; and we must suppose that it will modify the vital properties of these parts more than of others; there will be a spasmodic abruptness in the action of the muscular fibres there, or a permanent contraction of those fibres at the arterial orifices which are endowed with tonicity*; besides more or less thickening from the effusion of serum and soft lymph into the looser parts, as between the layers of the valves. These changes of properties vary with the progress of the disease. If the inflammation have continued for some length of time, the over excitement of the contractile fibres leads to their exhaustion, weakness, relaxation, and ultimately to a change in their structure; and the softening and effusions may lead to stretching, ulceration, rupture, or in time to harder thickening, contraction, and adhesion together of the looser and more membranous parts.

This brief sketch of the pathological effects of endocarditis, on which, had we time, we might enlarge with advantage, may suffice to render intelligible to you its physical signs, which I think I may describe to you with some confidence, having narrowly examined them in a good many cases, and traced them in a few to the conditions disclosed after death. They are often combined with those of pericarditis, which may come on after or before them, and always very much mask them. Like in pericarditis, the pulsations are not always more frequent in the first instance, but the sounds are louder and the impulse is stronger. In a few examples this has been all, and the loudness and strength have in the course of a few days subsided. But more generally the first sound becomes prolonged and double, or attended with a slight roughness, and eventually lengthened out into a blowing or grating murmur. This announces a modification in the current of the blood in some part of the heart, and if you attend to the character of the sound and the situation where it may be best heard, you may with tolerable precision determine in which part of the interior of the heart it is produced. If it is heard best an inch or two below the left breast, where the apex beats, and is heard but little or not at all above the upper half of the sternum and in the carotid arteries, it is produced by regurgitation through the mitral valve, the closure of which the irregular spasmodic action

* Arteries gradually contract on the application of a mechanical or chemical irritant to them. It is most probable, therefore, that the fibres, in which this tonic contractility resides, would be more irritable and contract more under the first influence of inflammation. Our Committee have been making experiments on this point; the results are interesting, but not yet complete.

of the fleshy columns renders imperfect. I before drew your attention to the nicely adjusted mechanism of this valve ; how, for its closure, there must be an equal drawing of all its cords, to spread the membranous laminæ against which the blood presses ; and you can now see how either the excessive or the defective drawing of some by the spasmodic, or by the weakened contraction of their fleshy columns, would throw these laminæ into wrinkles or loops, forming little chinks, through which the blood may be squeezed in a sonorous jet at each contraction of the ventricle.

If, again, the murmur is distinct along the upper half of the sternum and in the carotid arteries, where it commonly has a rougher, more grating character, you may conclude that it is produced in the aortic orifice. It implies the presence of a partial obstruction to the current, formed by swelling or deposit of lymph on the valves, or by the tonic contraction of certain fibres, all results of the partial endocarditis. In these cases, the clear flap, which constitutes the second sound, is often also impaired, being dull or double ; and is sometimes accompanied, or even replaced, by a short grating sound, which is heard best about the middle of the sternum, and may sometimes also be heard in the carotid arteries. These altered sounds arise from changes in the arterial valves, which, being thickened by deposit on them, or by infiltration between their laminæ, cannot flap clearly, and may not perfectly close, but leave a little chink between their edges, through which the blood regurgitates with a slight grating noise on the diastole of the ventricles.

There are few cases of acute rheumatism which do not, during their course, present some of these signs, which sometimes are transient, and cease with the fever, but more commonly continue for a long time after, and often become permanent. As far as my observation goes, I should state that those indicating the affection of the aortic orifice are less enduring than those of the mitral orifice ; but, whilst they last, they are commonly attended with more constitutional disturbance, and give to the pulse a sharp jerking character. It probably requires a more considerable lesion in this orifice to produce sounds than in the mitral orifice. In the latter situation, the properties regulating the action of the valves are so readily deranged, both by inflammation and by its effects, that the blowing murmur which is heard there often becomes more or less constant, or is produced whenever the circulation is accelerated. The degree in which the regurgitation may prove injurious, will chiefly depend on its extent and on the state of the general circulation. If the heart's propulsive power be weak, or the regurgitation considerable, which may often be known by the lower or deeper tone of the murmur, there will generally be more or less dyspnœa, especially on lying down or on lying on the left side, sometimes with feeling of faintness or palpitation, and perhaps cough ; palpitation always on exertion ; sometimes an unequal or irregular pulse ; and occasionally more or less pain in the left side. But if the action of the heart is pretty effective, and the regurgitation slight, which may be known by the more whif-

ing or whistling character of the murmur below the breast, there may be little or none of any of these symptoms. But almost in all cases the regurgitation will induce secondary effects, by slightly but constantly *backening* the venous circulation, and causing gradual congestion in many viscera, which may more or less derange their functions, according to their proneness to disorder. Hence occasional gastric and hepatic derangements, or "bilious attacks," from accumulating congestions in the portal system; these are very common: attacks of asthma and pituitous catarrh, from pulmonary congestion; and attacks of headache, drowsiness, or giddiness, from stagnation of blood in the sinuses of the brain

Thus you have in endocarditis many of the signs and pathological effects of valvular disease, and for this obvious reason, that this inflammation constitutes or induces valvular disease in its several varieties, and either in an acute or in a chronic form is its principal cause. So also endocarditis is often excited during the course of valvular disease or other organic lesions of the heart, causing considerable aggravation of their symptoms, generally increasing them, and hastening their fatal termination. We shall have to notice these effects under the head of diseases of the valves.

I shall say but little on the subject of *carditis* proper, or inflammation of the substance of the heart; for I am not sure that I have seen it, at least distinct from the membranous affections. In cases of pericarditis and endocarditis with disease of the valves, I have not unfrequently found portions of the muscular tissue of a darker colour and more or less softened, which probably is the effect of inflammation; but I am by no means confident of this. In one case in which I saw this livid red softening it occupied a considerable part of the posterior wall of the left ventricle: there was disease of the mitral valve, and thickening and redness of the endocardium near it. The patient had long suffered from palpitation, which had become aggravated of late, and his death was caused by spontaneous gangrene of one of the legs. A few instances are on record by Corvisart, Latham, and others, of pus being found between the muscular fibres; and Bouillaud describes other conditions which he supposes to be the effect of inflammation, especially a grey and a yellow softening. It appears to me probable that inflammation may lead to these conditions in some instances; but Dr. Copland has seen them (and so have I) in various cachectic states of the body, where there had been no signs of inflammation, but where all the tissues were more or less discoloured, the blood being apparently in a diseased state. To judge that the tissue of the heart is especially diseased, we must see that it differs much in appearance from the other muscles of the same subject. You will find, on comparing the same muscles in different subjects, a remarkable variety of colour; and in some there is no freshness in any of the muscles, but all are pale, and verging on a pinkish drab or dingy brick-colour. Where this is not the case, a pallid yellowish appearance of the substance of the heart is not at all an uncommon accompaniment of other lesions of the organ, especially adhesions of the peri-

cardium and accumulations of fat ; but I should be inclined to refer this to an altered state of the nutrition of the organ, owing perhaps to partial obstructions in the coronary vessels, rather than to the immediate influence of inflammation.

I can say nothing from experience on the symptoms and signs of carditis ; in the cases on record there have been described, pain in the region of the heart, with fever, dyspnœa, extreme anxiety, agitation, and perhaps delirium ; rigors, palpitation violent at first, passing afterwards into fluttering, fainting, and irregular movements ; and in a very few days death ensues. In simple and general carditis I presume the sounds would be altered in intensity rather than in character ; being louder at first and soon becoming weak : but it is probable that generally some parts would be more affected than others, or the apparatus of the valves would be also involved ; in which case there might be produced murmurs of various kinds. It is scarcely possible that acute inflammation can continue in a considerable part of the substance of the heart without interrupting its function, and rapidly destroying life ; but it is conceivable that a small portion might exist, and even form an abscess, or, in a more chronic form, lead to a change of structure. Whether the yellow fibrous transformation of the muscular substance that is occasionally met with be the result of a process of this kind, I have not had a sufficient number of facts to enable me to determine ; but I have seen, in cases of long continued disease of the heart, among other changes in which an inflammatory process could be recognized, a development of whitish fibres, and even laminæ, in the fleshy substance, which had much the appearance of the commencement of fibrous degeneration.

I shall return to this subject under the head of Hypertrophy.

LECTURE XXVIII.

Structural Diseases of the Heart.—Hypertrophy and Dilatation ; Causes.—Anatomical Characters of Hypertrophy.—Varieties and Combinations.—Pathological Effects.—Its relation to other Lesions.—Symptoms of Hypertrophy.—Physical Signs.—Variations of the Signs.—Dilatation of the Heart ; Causes.—Anatomical Characters.—Pathological Effects and Symptoms.—Physical Signs.—Dilatation of the Auricles.

Our next subject is the diseases of the *structure* of the heart. The shortness of time which we have left will prevent me from entering into much detail on this subject, which I regret the less since it has been treated by recent authors more fully than the functional affections. You will find, moreover, that many of the descriptions already given of functional disorder will apply also to cases of organic disease, for I have purposely avoided attempting complete distinctions between these, until they should appear from a rational examination of the nature of the two classes. When we speak of the symptoms of organic disease of the heart, we include the phenomena of its function, disordered in consequence of an injured mechanism or an altered tissue ; these as far as regards their physiological relations, may in great part be also produced by other causes less permanent than those of diseased structure ; and it is only in case of their great extent or permanency that they can be judged to indicate organic disease. You will not be surprised, therefore, if I do not dwell much on the general symptoms of organic diseases of the heart ; they are those of the several functional disorders that I have already noticed—increased, irregular, or diminished action, with variously modified sensibility—in addition to a few others, and to physical signs, which depend more directly and essentially on the particular lesions of the mechanism of the organ.

I shall occupy your attention, first, with diseases affecting the muscular walls of the heart ; and afterwards with those of the valves and the great vessels.

The *muscular substance* of the heart may become variously altered in quantity, form, and quality ; and its several changes may in different degrees be traced to causes affecting either the motory functions of the organ or its nutrition, or (as is more commonly the case) both together. The most common and familiar of these affections, hypertrophy and dilatation, opposite as they seem, often arise from similar causes, but operating on a different condition and quality of tissue. When from any cause, whether the quantity or quality of the blood or the irritability of the fibres, the heart is excited to excessive action for a considerable length of time, and the nutrient function of its vessels is not impaired by a general cachectic or chlorotic state, like any other highly exercised muscle it increases

in substance, it becomes over-nourished or *hypertrophied*: and there is, in the case of the heart, an additional reason why increased action should eventually lead to hypertrophy—namely, the direct relation of its nutrition to its own action. When speaking of inordinate action, I had occasion to observe that the great force of the pulsations is commonly expended on the first parts of the arterial tube; so that whilst the throbbing is strong in the aorta and immediate branches, the pulse in the radial and other distant arteries is often uncommonly weak. Now the coronary arteries, which supply the heart, are the first to profit by this partial force; and whilst they are furnishing the tissues of the heart with the nutrient fluid, in force and abundance greater than usual, distant parts may be languishing for lack of a due supply. Thus increased action may extend from function to structure, and continued excitement becomes perpetuated by augmented substance and strength. But if the blood be defective in its nutrient quality, in respect to muscular fibre, as it is in various cachectic conditions of the body, or if the strength of the heart itself be unequal to propel the mass of blood on which it endeavours to contract, excited action will not be backed by increased nourishment; the contractions, although still quick and abrupt, will have no extent of force, and the fibres, unable to expel the mass of blood to the usual amount, will become permanently extended—spread abroad: the walls will be thus *dilated*, and the cavities enlarged. There are other causes of dilatation, which we shall consider presently. Again, these conditions may be combined: as when the nutrition of the heart is augmented with the increased action, but still its force is inadequate to its task, and the walls suffer distension at the same time that they are over-nourished. This brief sketch of the pathological causes of hypertrophy and dilatation will render intelligible to you the circumstances in which they occur. The exciting causes of hypertrophy are habitual muscular exertion of an extreme kind, especially during growth, various diseases of the heart, both functional, increasing its irritability, and structural, exciting its action: these include rheumatism and other inflammatory and febrile diseases; various circumstances impeding the circulation and taxing the heart with increased labour—such as obesity, tumours, tense pulmonary emphysema, which compress the great vessels, and so forth.

There are a good many anatomical varieties of *hypertrophy* of the heart. It may be general or partial, accompanied by enlargement, or by diminution of the cavities. The size of the natural heart was stated, by Laennec, to be generally about that of the closed fist of the subject. This standard of comparison is more convenient than accurate; but unless we come to weights and measures, not of the heart only, but of the body in relation to it, we cannot readily get a better. This is a fit subject for the application of the numerical method by those who have time and opportunities for making it; and I have no doubt that interesting results might be

obtained from it.* In the meantime we must be content with ocular and manual measurement—the “rule of eye and thumb,” or rather of *fist* (I do not mean *pugilistic government*). You should, however, accustom yourselves to observe the position and dimensions of the heart, and the relative proportions of its walls and cavities, in every subject that you see opened, and you will soon get a tolerable notion of their healthy average in relation to the body. If you are inclined to be more accurate, I may refer you to the work of M. Bouillaud, who has given the measures and weights of a great many hearts. The following are about the averages which he gives of the chief measurements:—Circumference around the base of the ventricle, $8\frac{3}{4}$ inches; length, $3\frac{3}{4}$ inches; breadth, $2\frac{3}{4}$ inches; thickness of walls of left ventricle, $\frac{1}{2}$ inch; of right, $\frac{1}{4}$ inch; of the septum, nearly an inch, (this I think two much by $\frac{1}{4}$ th;) weight, between 9 and 10 ounces.

I believe these are pretty accurate measurements for adults, but they must be taken only as averages, subject to fluctuations somewhat above them and below them in varieties of size and age.

In cases of hypertrophy the heart may present various forms and degrees of enlargement, even to three times its natural size and weight. Lobstein has described an enlarged heart which weighed 32 ounces. Where the enlargement is considerable, there is always dilatation with the thickening of the walls. Here I would warn you against the common mistake of using the word hypertrophy as synonymous with thickening. There may be hypertrophy to a great extent without thickening; as where the cavities are much enlarged, and the walls therefore extended. The word hypertrophy means increase of substance, and may be very well applied to the whole heart or to an integral part of it, but not to express the increase in thickness, which is in one direction only. For the same reason I am not well satisfied with the names given by M. Bertin to the varieties of hypertrophy, for they fix the attention too much on the increased nourishment, and too little on the distending influence concerned in their modification. In these drawings and plates you see exhibited the varieties of hypertrophy. Here is *simple hypertrophy*, the walls being thickened, but the cavity natural. Here is *hypertrophy with dilatation*, the “eccentric hypertrophy” of Bertin. You see by these two examples that the walls may be either thicker or not thicker than natural. It is the active aneurism of Corvisart: I would, for brevity sake, call it *dilated hypertrophy*. Here you have its converse, *hypertrophy with diminution of the cavity*, which Bertin calls “concentric hypertrophy:” it may as

* Since these lectures were delivered, two valuable contributions to this department have been made by M. Bizot (Mém. de la Soc. Méd. d'Observation) and Dr. Clendinning (MEDICAL GAZETTE for June 9th, *et seq.*) The researches of these careful observers have established, by numerical proof, several notions to which I had been led by general observation, and which are noticed in the lectures. There are other interesting deductions which arise from these researches, which I may have occasion to notice elsewhere.

well be termed *contracted hypertrophy*, which in some measure expresses the cause of the diminution of the cavity.

These several forms of hypertrophy generally affect chiefly one of the compartments of the heart, and none so frequently as the left ventricle; next the right ventricle, and then the auricles. In the latter, dilatation is almost always combined with the hypertrophy. Dilated hypertrophy is, in fact, the most common form in the ventricles also. It is by no means uncommon to see particular parts of a compartment more enlarged than others. Thus the fleshy pillars of the mitral valve, those of the tricuspid valve, the cross muscular stays and net-work of the interior of the right ventricle, and the *musculi pectinati* of the right auricle, are frequently developed to an unusual degree. The increase in the pillars of the valves is generally associated with some defect of the valve. I have occasionally seen the thickening of the walls near the base much greater than near the apex; probably from the undue development of the fibres that encircle this part; rarely the converse is the case. In dilated hypertrophy of the ventricles there is a considerable difference in the shape which it assumes in different cases, the most remarkable varieties being those of elongation and lateral enlargement. Hypertrophy of the left ventricle, with elongated dilatation, is most commonly associated with disease of the aorta or its valves, especially those which permit regurgitation. In disease of the mitral valves or orifice the dilatation is usually more lateral or globular; but this form is met with also without valvular disease. In the right ventricle, also, the enlargement is in some cases more in the pulmonary, in others more in the inferior or auricular, portion of the ventricle; but I do not know that I can associate these differences with other particular lesions.

The muscular tissue in hypertrophy is not only increased, but it is often more or less changed. In the more sthenic forms, which are associated with active nutrition and a rich state of the blood, it is generally redder and firmer than usual. In leucophlegmatic or cachectic subjects, on the other hand, the increase of substance is often accompanied with duller colour and a laxity and softness which render the texture flabby, and probably cause its being, as it always is in such cases, dilated. In cases of organic disease of long standing, especially with close adhesion of the pericardium, I have repeatedly seen threads or laminæ of a dirty white tissue among the muscular fibres, many of these fibres having also partially lost their colour, and the others being in a flabby state. In other cases, the enlargement of the heart has been in great measure made up of a soft texture containing much fat, like that often found in the pelvis of the kidney. In rare instances, again, whole portions of the ventricles have been found transformed into a dense fibrous tissue. It is obvious that such changes must have an effect on the function of the organ, very different from that arising from simple increase of the muscular fibres. Yet it is not unlikely that they

may both originate from inordinate action of the organ, and the modified circulation which it causes through its own vessels, with different conditions of the nutrient fluid. We have no time for more on this subject, which is too speculative in its present state. I return to muscular hypertrophy.

The pathological effects of hypertrophy will necessarily vary according to its degree, the part which it affects, and the other lesions with which it may be complicated. The commonest and most important form is that affecting the left ventricle, and manifesting its effects on the general circulation and its functions. If the hypertrophy predominate, and be not counteracted by any valvular defect, there will be an increased strength of the arterial pulse, which will commonly be felt most in the arteries nearest to the heart, but it may extend to the whole arterial system. You can readily understand that the increased force of the arterial current may occasion various disorders of function and structure in the several viscera and tissues of the body. In the first place, it may cause dilatation and other changes in the coats of the arteries, especially those that more immediately feel its force, the ascending portion and arch of the aorta. Then as conducted into various tissues and organs, it may excite and disturb their functions, exalt their sensibility, and especially dispose them to inflammation, serous effusion, and hemorrhage, or aggravate any inflammations, irritations, or hemorrhages, when they occur. Hence apoplexy and phrenitis, epistaxis, ophthalmia, and other inflammatory affections of the parts of the body, have been traced to this form of disease of the heart. In time, the strong pulse accompanying hypertrophy of the left ventricle may cause an increased or modified deposition of nutriment in the different tissues which it reaches, particularly the parenchyma of viscera. The kidneys afford the best illustration of this, because they receive their blood only from the arterial system. In hypertrophy of any standing they are generally found enlarged, and otherwise diseased, and often presenting the granular albuminous deposit which has been described by Dr. Bright. The lungs and the liver are also very commonly increased in substance; but this must be in many instances partly referred to the obstructed venous circulation which so frequently accompanies hypertrophy of the left ventricle; being another consequence of its cause. The modification of nutrition especially affects the vascular system, whence arises thickening or ossification of the coats of the small arteries of the brain; and in the rupture of these there appears another mode in which hypertrophy of the heart may lead to apoplexy.

Hypertrophy of the right ventricle is commonly supposed to be a cause of congestions, inflammation, and hemorrhage, in the pulmonary tissues; but as these effects are seldom observed where there is not also some cause of obstruction to the onward flow of blood through the left side of the heart, it is uncertain what share the hypertrophied right ventricle may have in producing them. In fact, if you remember that its auricular valve is not formed to close

completely on an accumulating mass of blood, but permits regurgitation when the ventricle is distended, you can conceive that the pulmonary textures are not likely to suffer from its pulsations, unless their vessels are first distended by some other obstructing cause in or beyond them. In that case the force of the right ventricle must be felt; and when it becomes so much distended as to open its auricular valves, this force is then in part expended backwards into the auricle and veins, causing the venous swelling and pulsation often conspicuous in the jugulars. This venous obstruction, when considerable or permanent, leads to other effects, such as congestions in the portal system and in the sinuses of the brain; whence arise hepatic and gastric disorders, headaches, cerebral oppression, and apoplexy, and especially dropsical effusions of various kinds. These backward effects are the more likely to ensue when the right ventricle is much dilated as well as hypertrophied; whereas the effects on the pulmonary textures, congestion, hemorrhage, hypertrophy, and excessive bronchial secretion, depend rather on the increased strength, without much enlargement of the ventricle.

When the auriculo-ventricular orifice is contracted, we occasionally find dilated hypertrophy of the right ventricle combined with contracted hypertrophy of the left. It has puzzled pathologists to account for this hypertrophy of the left ventricle, when its task must be diminished by its receiving less blood to propel from the left auricle. It has been attempted to explain it by the left ventricle feeling the obstacle at its own auricular orifice through the whole course of the arteries, capillaries, veins, the right side of the heart, and the pulmonary vessels; a round-about explanation truly, and one that supposes a degree of distension of all these parts that is very rarely seen. Then Dr. Copland urges this case as an argument for his favourite notion of an active expansion of the ventricles, which I cannot admit to be warranted by any physiological analogies. But how very simple is the true cause of this hypertrophy, if I understand it aright! Why should not the excitement of the whole heart dependent on the distension of all its other cavities produce increased action, and eventually increased growth of the left ventricle also? Is it possible that the same fibres which encircle both ventricles can be excited in one and not in the other? No, surely; the left ventricle, naturally the strongest and most active, is thus excited by sympathy or continuity of irritation; and when its walls become increased under this influence, the cavity must be contracted from the smallness of its contents.

I cannot tell you of any remarkable pathological consequence of hypertrophy or dilatation of the auricles: they are to be considered as effects more than as causes. The strength of their contraction is too trifling to produce any other effect than that of stimulating more powerfully their ventricles, which are apparently excited to contraction by their impulse. This influence is perhaps reciprocal in the case of the right auricle and ventricle; and the consecutive pulsations of the two propagated backwards is sometimes seen in a

remarkable double pulse in the jugular veins. The dilatation of the right auricle is sometimes enormous. I have seen it large enough to contain an ordinary-sized heart.

From the general pathological effects of hypertrophy of the heart you may infer the prevailing character of its symptoms. A forcible, at the same time often not a very effective, state of the circulation, with its phenomena, palpitation and throbbing, occasional flushing, and a strong hard pulse, manifesting its effects on particular organs, as by giddiness, palsy, or apoplexy in the brain; dyspnœa, asthma, with thin mucous secretion, or occasionally hemorrhage in the pulmonary tissues; gastric dyspepsia, gastrodynia, sickness, discoloured alvine secretions, and other symptoms of disorder in the stomach, liver, and bowels; albuminous, high-coloured, and often scanty urine, with accompanying gouty or dropsical symptoms, referrible to disordered functions of the kidneys; more or fewer of these may be variously grouped together in different cases, as frequent effects or symptoms of the more sthenic forms of hypertrophy of the heart. Some of these secondary effects may occur and be the subjects of complaint, when those of the immediate action of the organ, the cause of them all, are not sufficiently marked to attract attention. When dilatation is joined with it, there will be more weakness of the circulation, with consequent torpidity of the functions of the extreme vessels, manifesting itself in venous or visceral congestions, œdema, and coldness of the extremities, pains in the back and limbs, and the incapacity of the organ, will be greater; whence a pulse, unequal in force and often irregular in rhythm, severe attacks of palpitation and dyspnœa, may then present themselves, the disease is more manifestly one of the heart itself.

The physical signs of hypertrophy are intelligible and characteristic. The increased mass of the muscular fibres renders their act of contraction stronger than usual; hence the impulse is also strong. But the character of this impulse, as well as the sound which attends the contractions, will depend on the form which the hypertrophy has assumed. When it is a simple thickening of the walls without increase or diminution of the cavity, the impulse will be gradual and heaving as well as strong, both because thick muscles cannot contract so simply or abruptly as those that are thin, and because the enlarged size of the heart brings more of it in successive contact with the ribs. So, likewise, the first or systolic sound will be prolonged, but duller than usual, because the sounding transition of thick walls, from loose to tight, is less extensive, abrupt, and instantaneous, than when they are thin. I formerly illustrated this by the different sounds produced by suddenly stretching fabrics of different thickness, such as linen and sackcloth, or thin stuff and thick baize. With contracted hypertrophy, in which the cavity is smaller, and the walls therefore have less extent of motion, the diminution and dulness of the first sound in the region of the heart is still more remarkable; it may be quite destitute of the flap usually heard, whilst the impulse is remarkably heaving, as if the heart

swelled against the ribs at each contraction. It often happens that the sound is louder at the top of the sternum and in the carotids, than in the region of the heart. The second sound, as heard at the mid-sternum, may be as loud as usual. When, on the other hand, the hypertrophy is dilated, there are better conditions for generating sound; the walls and valves being loose and flabby, pass with greater abruptness into the tense state of contraction, and yield a louder sound: whilst the impulse, although strong, is more abrupt and in cases of extensive disease is followed by a motion of collapse, from the sudden falling back of the large heart into a state of passive looseness at the moment of diastole. Both sound and impulse will then be extended over a wider space than is natural; and when the disease is considerable, and unattended with an emphysematous state of the lung, there will be also dulness on percussion, more or less extensive according as the enlarged heart is in contact with the thoracic walls. The situation of this dulness, and of the impulse, will vary according to the form of the dilated hypertrophy and other circumstances which affect the position of the organ. In elongated enlargement the impulse is generally felt below its usual spot, between the fifth and sixth ribs, down to the seventh or eighth; and I have even felt it in the abdomen, below the margins of the ribs. The dulness reaches from that part upwards to the sternum. But constant, or even occasional distension of the abdomen, by any cause, which prevents the descent of the diaphragm, will make the enlarged heart take another position, by which its apex extends further to the left, and the dulness on percussion reaches from that point to the sternum, occupying the whole intra-mammary, and perhaps part of the mammary region. The same position of the heart, thus more horizontal than natural, may, perhaps, also be caused, as Dr. Hope has pointed out, by adhesions of the pericardium, which prevent the organ from enlarging downwards. Enlargement of the liver, distension of the stomach or colon, and dropsical effusions in the abdomen, are the most frequent causes of this lateral direction which enlargement of the heart often takes. Similar causes may also determine the displacement to be outwards, against the thoracic walls, occasioning them to project to the left of the sternum, in the manner described by Bouillaud. This is most remarkable in children, and in young persons with narrow chests. When the enlargement is more transverse or globular, the dulness on percussion, and the impulse, are higher, independently of the position of the diaphragm; and this is particularly the case when the right ventricle is also enlarged.

I have known the dulness on percussion and impulse to reach over two-thirds of the left front of the chest, and as high as the third rib. You can understand why, under these circumstances, the impulse should be of a different kind as well as more diffused than usual, being produced by the swelling *body* of the ventricle rather than by the striking apex, and is therefore also felt higher instead of lower in the chest.

There are many other varieties, of which I would rather give you the principles than the details, for our time is very limited, and the circumstances which modify physical diagnosis are too many ever to be known by those who do not learn it fundamentally. I have told you that naturally the left ventricle touches the walls of the chest only at the apex, and that the impulse felt on full expiration at the sternum is that of the right ventricle. But if the left ventricle be much enlarged, and the right not, its impulse may be felt as far as the sternum, the impulse and sounds of the right ventricle being at the sternum only, or transferred to the right of it and the epigastrium. Again, if the right ventricle be also enlarged, it will keep the left ventricle to the left, and from the walls of the chest, so that its stronger impulse may be but little felt, nearly the whole front being occupied by the dilated right ventricle. Hence the reason why much enlarged hearts are often not felt beating with the force that might be expected from the thickness of the walls of their left ventricle; hence, too, the rules given by Laennec and others, to find the signs of each ventricle at particular spots, fail in many cases. The position of the whole heart, and of its compartments, is changeable, and when altered by disease it may be so different as to be entirely shifted from the common *landmarks*. It is rather by relative than by absolute rules that we can rightly judge here. For instance, if an hypertrophied left ventricle encroaches on the region of the right, we may know still that it is the left, by finding the different kind of sound and impulse to the right of it, and no other kind to the left. When the heart is displaced to the left, the signs of the right ventricle are those in the lower as well as right part of the space in which the sounds and impulse are felt most, those of the left ventricle being above and to the left.

To a certain extent you may also judge the strength of the left ventricle by the loudness of the sounds and the strength of the pulse at the top of the sternum and in the carotid arteries, when they are not exaggerated by dilatation of the aorta. Hypertrophy of the right ventricle could not cause these signs, whatever increase of sound or impulse it may produce in the region of the heart itself. Again, when the pulse is strong, and the sounds loud at the top of the sternum and in the carotids, although there be no strong impulse to the left of the sternum, there is probably hypertrophy of the left ventricle, but it is displaced from the walls of the chest by a dilated right ventricle, expends its force on the more yielding tissue of the lung, and in slender persons may be actually felt in the left back. You must perceive, on the other hand, how tumours, or a consolidated lung, which push forward the heart or prevent its receding from the walls into the spongy lung, must greatly exaggerate the impulse of the heart, and may give the impression of extensive hypertrophy where little or none exists. I have seen such effects from aneurism of the descending aorta, from tuberculous or inflammatory consolidation, and tense emphysema of the inferior lobe of the lung, and from extensive effusion in the right pleura. For analogous reasons hyper-

trophy of the heart is more perceptible and distressing in persons with narrow or distorted chests than in others. Many individuals with broad deep chests have large hearts that give them little or no trouble, but would cause distressing symptoms in those of a slender make. Such persons, if they gradually grow fat, may still retain much activity and strength. It is an advantage to them to have a heart strong enough for their increased bulk, with room enough for it to work in ; their balance is preserved. But should their bulk be suddenly reduced, the heart may then be too strong for the smaller frame, and may evince symptoms of its disproportionate strength. When, instead of increase of the muscular tissue, the heart is enlarged by the deposition of fat around it, the sound is obtuse as in hypertrophy ; but the arterial pulse and cardiac impulse are weakened also, and the dulness is more at the sternum, or middle portion of the chest.

Dilatation of the heart is in many respects the converse of hypertrophy. It stands in the same relation to diminished force of action, as the latter affection does to increased action ; and although sometimes induced by similar causes of obstruction to the passage of blood from the cavities, yet there must be in this case an opposite condition of the muscular tissue, a weakness and disposition to yield, instead of the tendency to react on, and struggle against, the obstacle which leads to hypertrophy. Where this reactive tendency is still unable to overcome the obstacle, or where the cohesion of the tissue gradually yields in the struggle, the two conditions, hypertrophy and dilatation, are at the same time produced. A softening or diminished cohesion of the tissue, may in itself be a sufficient cause of dilatation. To be brief, the causation of dilatation may be summed up in this : when the heart is incapable of sufficiently expelling its contents, whether in consequence of obstruction in the vessels from it—of regurgitation into it through imperfect valves—of want of power, of irritability, or of both, it becomes distended, and in time permanently dilated. The circumstances which determine these conditions, that is, the occasional or exciting causes of dilatation, are weakening influences in general, such as those of long-continued fevers, cachectic states of the system, with bad blood ; and causes which weaken the heart in particular, such as continued inflammation, obstruction of the coronary vessels, and such circumstances which injure the nutrition of the organ.

The anatomical characters of dilatation are not only the thinning and extension of the walls of the affected compartment, but also generally a paler and more flaccid condition of their muscular fibres. In some parts, particularly of the auricles, and at the apex of the left ventricle, the attenuation of the walls has sometimes proceeded so far, that the pericardium and endocardium are in contact, and these are occasionally thickened by opaque deposit, as if to strengthen them at these parts. The right ventricle and the left auricle are the most common subjects of simple dilatation. In the other compartments it is occasionally met with, but more generally combined

with some degree of hypertrophy ; so that, although the walls be thinner than natural, their greater extent gives them an increase of substance. The right auricle and ventricle are sometimes dilated to an enormous extent, with thinning of their walls, but still increase of substance ; and this condition is generally found to be associated with disease of the mitral valve. Dilatation of the ventricles is commonly in all directions, rendering the cavities globular ; but it is occasionally partial, the walls being distended into a pouch or aneurism, which in rare instances attains a considerable size, and may end in rupture.* Some of these cases are curious, but we have no time to spare for them. With the dilated condition of the walls there may appear various other traces of disease in the lining membrane and valves ; such as opacity, thickening, and roughness. They are most seen near the valves and orifices, and are evidently the result of an inflammatory process, which may have been either the cause or the effect of the dilatation. The orifices and their valves are commonly somewhat dilated, as well as the other walls, so that they may still maintain a sufficient proportion to perform their office. The dilatation of the semilunar valves is sometimes considerable, and renders them so thin that the fibrous threads can be seen forming an irregular network in them. Their thinning sometimes amounts to perforation, especially at the margins which apply to each other, and then this fibrous network may be the only part left. The tricuspid valve is seldom expanded in proportion to its orifice, which almost always partakes of the dilatation of its ventricle ; hence there is free regurgitation through this orifice.

The pathological effects and symptoms of a dilated heart are such as we have already described as those of irregular and defective action. Having here become an effect of permanent change of structure, they are necessarily more enduring than where they are merely functional ; and there are added to them others, arising from this more permanent influence on the other structures and functions of the body. When the left ventricle is dilated, its propulsive power is impaired ; there may be faintings, alternated with palpitation and other irregularity ; the whole systemic circulation is languid, and causes the symptoms of muscular debility, coldness of the extremities, defective and disordered secretions, imperfect nutrition, and eventually œdema of distant parts. When the change is chiefly in the right ventricle, the symptoms are those of impeded venous circulation, lividity of parts naturally red ; anasarca, often not confined to the lower extremities, perhaps with ascites and general congestion of the abdominal viscera. To these are often added the symptoms of pulmonary congestion, with its distressing concomitants, asthma, cough, profuse expectoration, and hæmoptysis, induced by the same cause which led to the dilatation of the right ventricle—disease in the mitral orifice. The complication of dila-

* A very complete memoir on Partial Dilatations and Aneurisms of the Heart, by my friend, Mr. Thurnam, has lately appeared in the *Medico-Chirurgical Transactions*.

tation with valvular disease is particularly unfavourable, for it constitutes a double defect in the power of the organ, whilst its irritability may continually excite it to fruitless and most harassing efforts of palpitation, which may terminate life by ending in syncope, or wear it out in a more gradual manner.

The physical signs of dilatation were well described by Laennec ; they are the converse of those of hypertrophy, and are equally intelligible and characteristic. Extended into a thin layer of muscle, the contractions of the walls are simple, abrupt, and quick ; and thus, although they have little power, they are well calculated to produce a loud sound and slight brisk impulse ; and these are diffused over a wide space, proportioned to the extent of the dilatation and its proximity to the walls of the chest ; but the sound can have little duration, and the impulse little strength : and even under the influence of palpitation do the pulsations not raise the walls, as an hypertrophied or even a healthy heart does when excited. The first sound of a dilated heart so closely resembles the second, that it is not easy to distinguish between them without placing the hand at the same time on the region of the heart, or on one of the carotids, to find which sound corresponds with the pulse, I should express this sound by the word *lup-tup*, instead of the *lubb-dup* of the healthy heart. The sound on percussion will be also dull in the region of the heart, in some proportion to the extent of the disease ; more extensively so than is usual in simple hypertrophy, but less than in dilated hypertrophy, in which the greater size to which the organ attains, and its flaccidity, which exists as in dilatation, brings a larger surface in apposition to the walls of the chest. To a certain degree we may, by the position of these signs, determine which compartment of the heart is most dilated, those of the left ventricle being, as Laennec pointed out, situated between the cartilages of the fifth and seventh ribs, and those of the right at the lower half of the sternum, and in the epigastrium. A dilated right ventricle, however, sometimes extends far to the left of the sternum, carrying the left ventricle behind and above it away from the walls of the chest, which, as in dilated hypertrophy, may prevent the impulse and sounds of the latter from being readily distinguished from those of the right. Under these circumstances a complete expiration, and leaning forwards to the left, will sometimes bring the heart into more complete contact with the walls of the chest, and give the stronger impulse and duller sound somewhere under the left breast. On the other hand, when the left ventricle is dilated, the impulse is felt lower than usual ; but it does not displace materially the position of the right ventricle, which may be heard and felt at the sternum. In consequence of their greater loudness, and the large size of the body in which they are produced, the sounds are heard over a greater extent of the chest than usual ; but I cannot agree with Laennec that this extent is so perceptibly proportioned to the degree of the dilatation as to be an exact measure of it. The transmission of the sound through the lungs may be so much modified

by even temporary conditions of the lungs (and much more so by consolidation, hypertrophy, or emphysema,) that the distinctness of the sounds in different parts of the chest depends as much on these organs as on the intensity of the sounds themselves.

Extreme dilatation of the auricle, particularly the right, may cause dulness on percussion at the middle of the sternum, and on either side of it; in fact, it only adds to the extent of the dulness produced by the enlargement of the ventricle which is conjoined with it. Laennec taught that dilatation of the auricles increased the loudness of the second sound; and although his view of the cause of this is totally erroneous, I think I can from experience confirm the fact, that the second sound has been sometimes unusually distinct at the middle of the sternum and on either side of it, in cases in which examination after death showed one or both of the auricles to be dilated. The second sound is, you know, produced by the flapping tension of both sets of semilunar valves; and although the dilated auricles which lie close to these valves do not increase the sound, they transmit it to the front wall of the chest better than the spongy tissue of the lung, which generally intervenes, could do. I once saw a double pulsation between the third and fourth right ribs, close to the sternum, which I was led to ascribe to a greatly dilated right auricle. The same double pulse was in the jugular veins; and I believe, with Laennec, very commonly attends extreme dilatation of the two right compartments of the heart, there being, in such a case, pretty free regurgitation through the auriculo-ventricular orifice.

In regard to the constitutional effects and symptoms of dilatation of the heart, I need say little in addition to what you have already heard of its origin and pathological tendencies. It is at once the effect and the cause of weakness in the compartments which it affects, and it extends the effects of this weakness by imperfect circulation of blood in the parts to which it supplies, or from which it receives, this fluid.

LECTURE XXIX.

Diseases of the Orifices and Valves of the Heart.—Anatomical Characters and Varieties.—Soft Thickening.—Tough Thickening; Contraction, &c.—Ossification.—Thickening with Ulceration, Vegetations, &c.—Atrophy of the Valves.—Prevalence of Disease on the left Side; its causes.—Pathological History and Physical Signs.—Principles of Diagnosis of Valvular Disease.—Disease of Aortic Valves; Signs; General Symptoms.—Disease of Mitral Valve; Signs; General Symptoms.—Disease of Pulmonary and Tricuspid Valves.—Diseases of the Aorta.—General Dilatation; Signs.—Aneurisms; Symptoms and Signs.

WE have seen a little of the effects which result from diseases of the fibres which move the blood; we have now to attend to the

chief lesions of that mechanism by which this motion is directed and conducted—the orifices, with their valves and great arteries.

Diseases of the orifices and their valves are of very considerable variety, but those that interfere with the office of the parts which they affect may be reduced to two classes—those that more or less *obstruct* the current of the blood in its proper channel, and those that occasion it to take a *reversed* direction. The former, for brevity, I term *obstructive*, the latter, *regurgitant* lesions. You now know so well the natural structure and function of the several valves, that the description of the anatomical characters of a few of these lesions will enable you to understand their effects in interfering with the mechanism of the circulation.

The most common change in the valves is *thickening*, which presents itself chiefly under two forms:—1. A *softer* kind of *thickening*, in which the valves retain much of their pliability; 2. An *opaque thickening*, with more or less *induration*, so that the valves become less flexible and mobile than usual. The first occasionally affects the semilunar valves, but rarely the auricular. It appears to be the product of inflammation chiefly affecting the serous membranes of the valves, and is produced by organized lymph between their layers, or upon their exterior, from which it often may be separated. This deposit is generally seen less at the margins than at the middle and attached parts of the semilunar valves, chiefly on their ventricular sides, and occasionally forming a bond of *adhesion* between two adjoining valves, which are glued together as far as the corpora arantii, as you see in these drawings. This constitutes a form of slight obstructive disease of the arterial orifice. I have in a few cases seen the same deposit on the layers and cords of the auricular valves, here and there causing them to adhere, and forming a false membrane on their auricular surface.

The *opaque tough thickening* of the valves is the most common, and is frequently combined with the former. When occurring simply, there is a smoothness of the exterior, which, with the character of the thickening material, seems to indicate that it is chiefly between the serous layers of the valves, and probably arises from disease in the fibrous tissue, that forms the strengthening web of the valves. This thickening commonly affects the semilunar valves to their very margins, and is sometimes seen extending into the ventricular lining, and perhaps forming there slight ridges where the bands of fibrous tissue cross the muscular walls. With the laminae of the auricular valves it commonly involves their orifices and part of the auricular lining, and extending into the tendinous cords, which are irregularly thick and knotty, and their fleshy columns are likewise occasionally changed into fibrous cords. With this more rigid thickening there is often combined a *contraction* or *elongation* of some parts, or both, affecting different parts; and it is such changes of proportion that so commonly injure the proper action of the valves and orifices. Mere thickening, unless it be attended with great stiffness, will not materially injure the office of the valves;

but changes in form and proportions necessarily must. Here you see several drawings illustrating these changes. In these affecting the mitral valve, you may perceive how the shortening of a few of the tendinous cords throws the rest into a loose state, in which they cannot draw the membranes of the valve smooth on each other; hence they form folds and chinks, through which the blood must regurgitate at each systole. Here, again, you see instances of portions of the valve and their cords elongated and enlarged, so that they may be forced backwards towards the orifice, and by keeping the valve partially open, also occasion regurgitation. Here again the two laminae of the valve are thickened, contracted, and adherent, forming a sort of narrow funnel-shaped tube instead of a valve, both retarding the flow of blood from the auricle into the ventricle, and never closing against its reflux into the auricle. This forms both obstructive and regurgitant disease of the mitral orifice. In this manner the left auricular orifice is sometimes so contracted as not to admit a little finger; naturally it admits with ease two full-sized fingers. Here again, in the aortic valves, you see one of them contracted at its free margin, so that it could not meet its fellows, but left a chink through which the blood regurgitated with a murmur into the ventricle at each diastole. I have known the same effect to arise from this kind of disease preventing the equal dilatation of the valves. When a ventricle with its orifice is dilated, the semilunar valves are generally dilated with them, so that the proportions are preserved; but if there exist in one or more of these valves a rigid thickening that will not yield, the dilatation of the orifice renders them insufficient; they cease to close the orifice completely, and consequently regurgitation takes place through them. Again, an uncommon elongation of the free margin or attachment of one or two of the semilunar valves, so that these margins, instead of being closed against their fellows, become retroverted under them, constitutes another form of regurgitant disease of the semilunar valves, of which I show you here several specimens. The valves appear to have given way generally at their attachments to the artery, perhaps from the yielding of its fibres and the formation of a slight pouch in it; but you see the effect is to destroy an attachment of two of the valves, which, instead of forming bags, are either retroverted, or contracted and thickened, on a line with the ventricular convexity of the other valve. In these cases probably rupture of some fibres may have at some time taken place, and accelerated the change; but it is always associated with more or less of the thickening of which I am now speaking.

To the thickening of the valves and orifices there is frequently added great induration and even ossification; and this alone may constitute disease, generally of the obstructive kind. As such, it often affects the aortic orifice, particularly at the attached portions of the valves, and at the curves of the artery, which form the boundary of the little recesses or sinuses into which they retire. If in either of these situations a rigid portion project into the current as it

passes, it constitutes obstructive disease, and may cause a constant murmur with each pulse ; or if the whole ring be rigid and unyielding, it may, under the circumstances of greatly increased action, constitute a constriction, the vibrating resistance of which, opposed to the current, may give a murmur. Sometimes the whole of the valve is so rigid that it must have opposed more seriously the passage of blood. In these drawings you see aortic valves which are ossified, and have adhered at their edges so as to leave only a little aperture at their middle, where their margins were loose and permitted the blood to pass.

The lesions which I have been describing are the most common forms of diseases of the valves ; they are mostly of a chronic character, and in the greatest number of instances originate in rheumatic endocarditis. Their effects on the circulation and on other functions have been generally proportioned to their degree and character, the rapidity of their production, and to their complication with hypertrophy and dilatation, or other disease : hence they may vary infinitely ; they may exist and produce cognizable signs, without obviously deranging the heart's action, or in an overt direct manner injuring the health ; and they may cause the most distressing and dangerous symptoms, and sooner or later prove fatal.

There is another kind of thickening to which the valves are subject, accompanied with softening, ulceration, and often rupture. This is fortunately not very common ; for it is a terrible disease, destroying the valves, chiefly the aortic, in the course of a few weeks ; and soon after proving fatal. This occasionally supervenes on older disease which has originated in rheumatism ; but I have in several instances known it to arise independently of rheumatism, perhaps after a severe cold, or violent strain, especially in persons who have been addicted to spirits. The ruptured or ulcerated portions of the valves are found loaded with ragged, soft, fragile vegetations, more or less tinged with blood, and these are also sometimes seen adhering to adjacent parts where the endocardium is entire. The remaining parts of the valves are much thickened, and opaque yellowish white, with a pink hue ; and pink patches are often seen in the aorta, with atheromatous thickening. I think that these changes may be viewed as the effects of acute inflammation affecting all the tissues of the valves. In these two drawings you see specimens of its ravages. In this, all three aortic valves have been completely broken up, and their torn margin fringed with thick vegetations hung down into the ventricle. In this, again, you see one of the valves has given way at its centre, and its margin, thickened and loaded with vegetations, lies like a cord across the mouth of the artery, whilst the lacerated attached portion is retroverted into the ventricle, and, with part of the lamina of the mitral valve near it, is covered with vegetations. In other cases I have seen smaller perforations, probably ulceration, both in the middle of the aortic and in the mitral valves ; always fringed with vegetations. The smooth perforations so common at the free margin of the semilunar valves

are of a different character, and not dependent on the same cause. I believe that in time the vegetations just described may become organized, forming the cartilaginous or fibrous little bodies which have been called warty excrescences, in which osseous matter is often found, and which generally have more recent vegetations attached to them.

Lastly, we have another class of valvular diseases that have been very commonly overlooked. I mean atrophy or wasting of the valves, by which their membranous portions, may become shortened or perforated, and the tendinous cords withered and absorbed away. The semilunar valves, both aortic and pulmonary, present these in the most obvious manner in the oval perforations, at their free margins. You see in these drawings several specimens. The perforations are oval or rounded, with their edges quite smooth and thin, as is also the whole valve. In fact, although there may be partial deposits of false membrane on their ventricular surface, the valves are most commonly very thin and flaccid when they present these perforations; and in other parts there may be seen still thinner spots that are separations of the fibrous web, and are all but through the serous membrane also. Now so long as these perforations are confined to so much of the margin of the valve as closes against that of its fellow, they may not produce regurgitation; and this is very commonly the case. In the mitral valve, the wasting, usually affects the posterior portion, the membrane of which is often annihilated by it, the cords being inserted directly into the auricular ring. The anterior lamina is also occasionally found much shortened, and without those fine thin expansions of membrane which commonly unite the cords to each other, below their insertion into the thicker part of the valve. It is pretty plain, that with this state of the valve, if there be not habitual regurgitation, inordinate action of the heart, or slightly disturbing circumstances, may induce it, especially if there be at the same time dilatation of the orifice. I have known a murmur produced by flatulent distension of the stomach, and by certain postures, which I have been inclined, for reasons to be explained afterwards, to refer to this kind of imperfection of the mitral valve. I have found these atrophied conditions of the valves in cases where there was no trace of previous inflammatory affections of the heart. In one case there was no other thoracic disease at all, the patient having died of fever; and attention was turned to the heart only in consequence of there having been many heart symptoms, with a constant blowing murmur below the left breast, during life. In some cases the smooth perforations have been found in valves thickened by inflammation, probably of more recent date.

With these several forms of valvular disease, especially those of an inflammatory origin, there are commonly combined various forms and degrees of hypertrophy and dilatation; the production of which may be more or less ascribed to and explained by the nature of the valvular disease. Thus hypertrophy with dilatation commonly

affects a ventricle the orifices of which are partially obstructed, or permit regurgitation, either of which conditions tends unduly to irritate and distend it; and as the efforts of the ventricle continue to be powerful or not, the hypertrophy or the dilatation will prevail.

You must have perceived from the descriptions already given, that the valves of the left side of the heart are far more commonly diseased than those of the right. Yet occasionally the latter do present the same changes as the left valves, but it is very rare that they are alone affected, or in a much greater degree than the left. Nor can you wonder at this when you consider the function, structure, and relations of the left side of the heart as compared with the right. Its function requires that it should be endowed with much greater strength, that it may propel the blood through the great circulation; and this greater strength exposes its parts to more violence from its own movements. Then, to bear this violence, its valves and orifices are furnished more abundantly with a strengthening fibrous tissue; yet this very tissue is obnoxious to inflammatory affections, which tend peculiarly to alter it. A tissue which protects by its strength cannot be endowed with high vitality; as its vitality is low, so is its natural reparatory power slow, and the hastening of this process by inflammation changes the nature and strength of the material. Hence there is deposited no longer the fine, even, transparent, capillary fibres, which only glisten into view with a silvery whiteness when they are bundled together in numbers, but an opaque, yellowish white, thick, tough material, partly fibrous and partly amorphous, possessing neither the strength nor the delicate flexibility of the original texture, and consequently injuring the apparatus by its bulk, its stiffness, or its liability to extension or contraction, laceration or rupture. Then the very perfectness of structure, that gives to the left ventricle a superiority in point of strength over the right, exposes it more to the effects of violence or excessive action. When the right ventricle becomes distended, you have seen that its auricular valve opens and permits a partial reflux; but the mitral valve of the left ventricle is made to close perfectly, and to bear the whole strain of the muscular fibres contracting on the blood. Again, the pulmonary valves are exposed to pressure from the tension of the pulmonary vessels only, which are exposed to little pressure but that of the expiratory forces; but the aortic valves receive the strain of the great arterial system, liable as this is to violent increase from the pressure of the muscles of the body, and of any sudden blow or impression on regions containing its larger branches. You will not wonder, then, that the valves of the left ventricle should be more frequently and more extensively diseased than those of the right. You have rather reason to wonder they suffer so little, and how they can stand so well and so long the strain to which they are exposed, particularly when disease has once begun in them. I believe that they do suffer much more frequently than is generally supposed, and that many of the circumstances which we noticed as causes of inordinate action of the heart,

do often irritate, or mechanically strain and injure the membrane covering the valves, and excite a temporary inflammation in it. How else are we to account for the partial thickening so commonly seen, especially in the aortic valves, even where there had been no history of any complaint particularly affecting them? That such a thickening has been slight and harmless we may ascribe to the perpetual motion of the valves and sweep of the current, which generally prevent any accumulation of deposit that is not connected with an intense inflammation, or one affecting the subserous tissues. But occasionally circumstances may favour an accumulation; thickening and rigidity may take place; and thus disease will now and then begin in the valves, independently of rheumatism or any other common cause, and gradually infringe on the integrity of their function. The atrophous form of disease, however, which produces the thinning and oval perforations in the semilunar valves, and wasting of the membranous parts and tendons of the auricular, is commonly met with in either compartment of the heart. It was on the right side that I have seen it attain the greatest degree, having reduced the membranes of the tricuspid to mere fringes, and the margins of the pulmonary valves to a network of threads. But even such disease on this side of the heart produces less prominent symptoms than slighter lesions on the other.

We shall now consider the pathological history of diseases of the valves; and as a distinction between them can scarcely be made without a study of their physical signs, we will begin with this subject, which will supply us with further knowledge of their more essential pathology. I have before had occasion to mention to you that the phenomena called murmurs are often produced by the modifications which valvular disease occasions in the current of the blood. In order that they be produced there must be the requisite elements, sufficient force in the current, and such a modification as may constitute a vibrating resistance to it. You can understand that valvular disease may be present without forming these elements, and therefore without any attendant murmur, as when the current is too languid, or the resistance too loose or yielding to cause vibrations; but in by far the greater proportion of cases, valvular disease does cause murmurs, either constantly, or under the circumstance of the current increased by exertion or other cause of excitement.

I hope that you now comprehend the principles of the production of sound too well to fall into the common error of supposing that the amount of a valvular lesion is indicated by the *loudness* or extraordinary character of the murmur. The loudest and most singular murmurs are the whistling, cooing, or musical, yet they are occasionally produced by comparatively slight lesions. The loudest musical murmur that I ever heard was produced by the retroversion of only the margin of one of the aortic valves, which you see in this drawing, the rest being in a pretty capable condition. The murmur was a loud cooing with the second sound; and the patient dying of fever, we had the opportunity of verifying the diagnosis which we

had made of the disease. Again, in this case, where you see all the aortic valves entirely broken down by disease, the murmur, which was double, was obscure and distant.

The *quality* of the murmur is a better index to the character of the lesion, but it is by no means a sure one. Uniform and shrill murmurs, whether blowing or musical, generally indicate slighter lesions than the rough or grating and deep-toned, because they are commonly produced where there is no great looseness or irregularity of the aperture through which the current generates the sound; they are most frequently caused by regurgitation through small smooth chinks between the mitral or the aortic valves. The rough grating or sawing sounds generally indicate more extensive disease, not being produced (as it has been asserted by others, and stated in my treatise on their authority) necessarily by indurated or osseous irregularities, but by a larger current passing over a body which offers to it an irregularly vibrating resistance. Such murmurs most commonly occur in the aortic orifice. It was a remarkably rough double sawing murmur which accompanied the lesion of the aortic valves represented here.* One of them is broken through all but its margin, which is left a ragged cord across the mouth of the aorta; there were here plenty of soft vegetations, but no material induration. A rough grating with the first sound was produced in this other case also, where the aortic valves were perforated by atrophy, leaving mere threads at parts of their free margins, which, forming loops, might catch and flutter in the current.†

The more distinguishing circumstances in regard to murmurs are, however, their relation to the natural sounds or motions of the heart, and the points at which they can be best heard. Laennec and Bertin paid some attention to these points, but without obtaining any general results. I believe that Dr. Hope was the first who attempted a diagnosis between different valvular lesions by the apparent situation of their murmurs; but this attempt was not founded on correct acoustic principles, and the means of distinction announced by him have failed when tried by others. He asserted that the disease of a particular valve may be known by the murmur being heard best at a spot on the walls of the chest nearest to that valve, and by its being whizzing or deep-toned, according to the proximity or remoteness of the valve from the walls of the chest. But only look at the anatomical position of the different valves. All four sets are comprised within a space of a couple of inches, being placed, in regard to the front of the chest, one behind another rather than side by side. Then see how the lung intervenes between them and the

* These and other illustrative drawings being too numerous to be introduced here, are reserved for a separate work. The notices of atrophy, polypous concretions, malignant diseases, textural transformations, malformations of the heart, and other subjects of minor import, are also omitted.

† In our experiments with India-rubber tubes, the best imitation which we could make of the sawing or rasping sounds, was produced by directing a strong current across rather loose threads or strings. This corresponds with the experience recorded in the lecture.

front wall of the chest, and must obscure and confound the sounds from beyond it; and you will acquit me of the imputation of stupidity when I tell you that I have never been able, in so small a space, to discriminate between the murmurs generated at the different orifices. But there are principles of diagnosis in these murmurs, and if they be carefully and rationally studied, they will generally enable us to distinguish, as far as is useful, between the different kinds of valvular lesion. They are nearly such as I gave them in the last edition of my little treatise on Diseases of the Chest;* and I have since been enabled to confirm and correct them by the examination of very many cases before and after death.

In judging of the seat and cause of a cardiac murmur, you have to attend chiefly to the *period* of the heart's motions at which it occurs, and the *manner* in which it is *transmitted* to the surface. A murmur accompanying only the first sound or impulse is necessarily caused by a current of blood *from* a ventricle; one accompanying, following, or replacing the second sound, must arise from a current into a ventricle. This simplifies the matter; but there are still two ventricles and two orifices to each; and how are the murmurs of these respectively to be distinguished? not by the different positions of these orifices certainly; they are too close together for that; but *by the different directions in which the sonorous currents spread the sounds, and the different manner in which they are transmitted to the walls of the chest.* Thus murmurs generated at the origin of the arteries will generally be more or less transmitted in the direction of the current along these arteries; and those produced in the auricular orifices will be conducted both by the current into the auricles, and by the tightened cords and fleshy columns to the apex of the heart, which is generally more or less in contact with the ribs. So far for the general principles: now let us consider briefly the special diagnosis of particular lesions.

Obstructive disease of the aortic orifice (observe, by *obstructive* I do not mean that the actual impediment is always much) is generally attended by a murmur with the first sound and impulse, heard in the region of the heart, along the upper half of the sternum, and in the right or both carotid arteries. The point where it is heard loudest will vary according to the position of the heart with regard to the lungs and walls of the chest. If it lie deep, and well covered with spongy lung, the murmur may be louder at the apex and in the right carotid than at any intermediate point, because the dense heart and the distended arteries convey the sound better than even a much shorter length of pulmonary tissue. In chests less deep, and especially where enlargement of the heart brings its base nearer to the sternum, the sound is loudest about the middle of this bone, or a little on either side of it; but still the more distinguishing character is, that it is heard above, in the direction of the innominate

* Since the publication of this work in 1835, similar diagnostic rules have been proposed by other authors, English and French, without due acknowledgement.

and carotid arteries, where sounds from the other valves can scarcely reach. The murmur is commonly like grating or sawing, and varies much in length and loudness in different cases. In some it lasts the whole period from the commencement of the systole to the occurrence of the second sound : in others it is a mere roughness or whizzing accompanying the natural sound, which may be pronounced *trrhuh-dup* or *djub-dup*. It is very often accompanied by regurgitant disease of the aortic valves ; and when this is not the case, the second sound (the *dup*) is frequently less clear than usual, which implies that the valves, from thickening, do not act freely.

Regurgitant disease of the *aortic orifice* produces a murmur with or instead of the second sound, by the sonorous gush of blood from the artery back into the ventricle. The place of greatest distinctness of this sound will also vary, as the former, according to the position of the heart and lungs : it is generally heard best at or near the middle of the sternum ; but its more characteristic feature is, that it obscures or supplants the second sound or flap at the top of the sternum and in the carotid arteries, which is usually very distinct there. The second sound of the right or pulmonary valves can often still be heard about the left margin of the mid-sternum. This murmur is seldom so rough or grating as that of obstructive disease ; for the current reflux from the artery is not so large or strong. It may, however, be very loud, and even musical, as in the case which I have lately mentioned to you ; and if the valves cannot close at any period of the diastole, it may even last from pulse to pulse. More commonly, however, it dies away before the next pulse ; and in slighter degrees of regurgitant disease, is very short, preceding and not supplanting the second sound. This is when the valves, before they are quite distended by the full recoil on them of the arterial column, permit a little jet of blood to grate between their margins, and make the second sound like *trrht* or *tzzt*, instead of *dup*. A little vegetation, or slight deposit of rough lymph on their margins, may cause this : I have often met with it both as a temporary and as a permanent phenomenon, when there was not much derangement of the heart's action.

Combined obstructive and regurgitant lesions of the *aortic orifice* produce the double, or to-and-fro sawing murmur, which is the succession of the forward and backward gushes of blood. They are by no means uncommon ; in fact, the murmur, instead of the second sound, indicating regurgitation, seldom exists without a murmur with the first also, indicating partial obstruction. These lesions, when at all considerable, are accompanied by dilated hypertrophy of the left ventricle, which often takes an elongated form, bringing its impulse lower and more to the left than usual. When the regurgitation is very free, it increases the force of the diastolic collapse so much, that I have known it resemble a second impulse. It also not unfrequently stimulates the ventricle to a second contraction, constituting a reduplication of the pulse : the rhythm otherwise is not commonly much affected. But the pulse generally

possesses a character that is remarkable. When the lesion is obstructive only, the pulse is generally hard and jarring; but when it is regurgitant also, each pulse, although momentarily hard and full, immediately recedes, which gives it a jerking or thrilling character. The same circumstance makes the pulsation of all the arteries visible, and sometimes locomotive,—a sign of lesions of the aortic valves first pointed out by Dr. Corrigan. I have seen it so extensive in an old man, that many arteries could be seen like worms under the skin, wriggling into tortuous lines at each pulse. The cause of this phenomenon is sufficiently intelligible. It proceeds from a defective equality of tension of the arterial coats. These vessels are distended at each pulse, and emptied the instant after, the aortic valves not, as usual, maintaining their tension; and if the coats of the arteries are defective in lateral elasticity, as in old people, they may admit each jerking jet of blood only by being elongated into a tortuous line. In its extreme degrees, and especially when existing in all states of the circulation, I think that this visible or moving pulsation of the arteries is pathognomonic of regurgitant disease of the aortic valves; but to a slight extent it may be observed in many cases of excited action of the heart, especially when there is a defective tension of the arteries, as after great losses of blood, and otherwise where there is a lax state of the vascular system. I should have pointed out to you a partial exhibition of this visible pulse as a sign of such a state of the arterial system. By raising the hand above the rest of the body, you will render visible the pulsations of the radial artery, which becomes partially emptied by gravitation, just as in case of aortic valvular disease the arteries in general become partially collapsed by regurgitation.

The general symptoms of disease of the aortic valves vary much according to its degree. When slight, it may occasion only more or less palpitation and shortness of breath on exertion, perhaps with a feeling of tightness or pain at the sternum, and other of the common symptoms of moderate disease of the heart. But when extensive, it is the most formidable kind of valvular disease, and generally proves fatal in the shortest time. I have known several cases run their course in a few weeks after the first complaint had been made of symptoms referred to the heart. It is probable that in some of these latent disease had been going on for some time in the valves, and was suddenly aggravated by mechanical violence or inflammation occurring, and to which alone the disease was ascribed. There then come on habitually increased action of the heart and dyspnoea, sometimes aggravated by fits of palpitation and orthopnoea, sometimes with angina, cough, and expectoration, often containing blood. There is commonly some anasarca, more rarely ascites and hydrothorax. Hæmoptysis and dropsical effusions have been too exclusively attributed to disease of the mitral valve and right side of the heart: I have seen them quite as frequently with severe aortic disease; in fact, where the left ventricle cannot effectually unload itself, whether from obstruction or regurgitation, there is just as much in-

interruption to the circulation, pulmonary and venous, as if the mitral orifice or the right heart were diseased ; and there is often a greater failure of the capillary circulation, from the impaired tension of the arteries. Among the effects of this, I may mention that regurgitant lesions of the aortic valves are often attended by a pallidity and emaciation, or in more acute cases by pasty puffiness of the integuments, with less colour than is commonly seen in mitral disease ; in which the symptoms are more simply those of venous congestion. There is often, too, a restless irritability about the subjects of aortic disease, comporting well with the jerking character of the pulse ; whereas with mitral disease it is not uncommon to see an unusual hebetude and torpor. But these distinctions are most uncertain.

Of the lesions of the *left auricular*, or *mitral valve*, we must now speak. *Obstructive disease of the mitral orifice* consists in its contraction, in the adhesion together of the laminæ of the valve and of their cords, or in bodies projecting into it. When this is capable of producing a murmur, it is at the time of the second sound, when the blood passes from the auricle to the ventricle at its diastole ; but I believe that a murmur thus caused is of very rare occurrence ; perhaps only in case of considerable contraction of the orifice with thickening of the walls of the ventricle, which gives them diastolic elasticity, and thus sucks the blood from the auricle with some force ; such was the state of the parts in one out of the only two or three cases which I ever met with, in which the murmur with the second sound was not distinctly referrible to regurgitant disease of the aortic valves. Where such a murmur does exist, it would probably be best heard in the same situation as the familiar one of regurgitant disease of the mitral orifice ; but its best distinction would be that it does not modify or supersede the second sound in the arteries, as regurgitant disease of the aortic valves does.

Regurgitant lesions of the mitral valve are attended by a murmur with the impulse and first sound, produced by a sonorous jet of blood through the imperfectly closed valve at each systole. I have more than once adverted to the nicely-adjusted mechanism of this valve, and I have given you a sufficient number of examples of the lesions by which this mechanism may be deranged. They are very common ; and my experience leads me to the conclusion, that of the instances of cardiac murmurs which present themselves in women and young persons below the age of twenty, five-sixths are caused by regurgitant disease of the mitral valve ; whilst in older persons, and those of the other sex, they are more frequently caused by disease of the aortic orifice. Now where may this murmur be best heard ? The mitral valve is situated about the cartilage of the fourth left rib near the sternum ; but the spongy lung, and the right ventricle too, if it be dilated, intervenes between it and the walls of the chest ; hence the murmur is seldom transmitted opposite to the valve so well as through the apex, to which the fleshy columns of the valves are attached, and which, at the time that the murmur is produced, is in close contact with the walls of the chest, some-

where between the fourth and seventh ribs below, or a little in front of the left mammilla. Here accordingly I have generally found mitral murmurs most distinct, being often quite superficial, and so loud as to eclipse the natural double sound; and this being still audible at the upper and lower ends and to the right of the sternum, and in the carotid arteries, where the murmur is inaudible or distant, completes the diagnosis. Great enlargement of the heart, or consolidation of the lung, sometimes transmits the murmur loud to walls higher up, in which case the dulness on percussion at the spot would explain the unusual circumstance. In a few instances I have heard a mitral murmur very loud and superficial about the third rib near the mammilla, and I have been led to suspect that it is in these cases transmitted by the dilated auricle which receives the reflux jet of blood. It is sometimes also very audible in the left back, and below the axilla. The character of mitral murmurs is generally blowing, but sometimes passing into a whistle, and more rarely grating. In some cases it accompanies the whole systolic act, and terminates with the second sound: in others it is confined to the end of the first sound, giving to it an additional vowel or a roughness, as we may express by the words *loo-dup* or *lurr-dup*; or, if confined to the beginning, *rehub-dup* or *jub-dup*. You may be amused at my new words; but I think that when your ears shall have become practiced, you will admit I have spoken truly the *language of the heart*. These murmurs are not always constant, especially when the action of the heart is irregular or unequal. We then often hear some of the beats with the murmur and others without it. Sometimes the murmur alternates with a double first sound, which I have compared to the footsteps of a cantering horse, and which I have conjectured to depend on a loose state of part of the mitral valve. When you once understand the principles of the production of these sounds, such varieties are intelligible enough.

As there is in most cases of considerable disease of the mitral valve more or less hypertrophy and dilatation of the heart, the signs of these conditions may be added to those of the valvular lesion. But if the regurgitation is free, it may much diminish the loud sound of dilatation, and the strong impulse of hypertrophy of the left ventricle, by removing the resistance offered by the closure of the mitral valve. Hence the tightening flap of the ventricular sound may be diminished as well as disguised by the murmur; and the strength of the impulse may be partially lost backwards into the auricle, and followed by a heavy diastolic collapse, when the blood flows in again from the distended auricle. When there is contraction of the mitral orifice there is usually more hypertrophy than dilatation in the left ventricle, with a strong impulse over it, and the blowing of regurgitation, which is scarcely ever absent, even in these cases. The other compartments are dilated, and may give, as usual, the short double sound.

The pulse varies very much in mitral disease. In many cases, particularly those in which there is constriction of the orifice, it is

very irregular and unequal, exhibiting the different varieties which I have described under the head of Irregular Action of the Heart (see Lecture XXVI.). It is generally weak also, particularly where there is either much constriction or free regurgitation. Yet I have known cases in which the murmur at the apex, during life, and the appearances after death, showed that the regurgitation must have been free, and the pulse was nevertheless strong and jerking, there being also dilated hypertrophy. And when the heart acts irregularly, the strong beats of the pulse are in some instances attended with a louder murmur at the heart than usual. In other instances again, where the pulse was intermittent, I have observed each intermission or weaker beat to be accompanied by a loud blowing at the apex, which was much less distinct in the other beats. This intermission was obviously from the force lost backwards in regurgitation, the cause of which was probably moveable, or subject to variation, as in the case of a partial looseness causing retroversion, or of an excrescence between the laminæ.

The general symptoms of mitral disease, both obstructive and regurgitant, are, besides those of inordinate and irregular action, those especially of pulmonary and venous congestion. Sometimes the disease appears to be more pulmonary than cardiac, the patient being asthmatic in paroxysms, or suffering habitually from dyspnœa, with cough, and copious thin mucous expectoration sometimes containing blood. In other cases, especially those of a slighter kind, the lungs suffer little, and the chief symptoms arise from a congested state of the venous system; such are enlargement, pain, and deranged action of the liver, hæmorrhoids, scanty and disordered urine, ascites, hydrocele, and anasarca, congestive headaches, giddiness, &c. These occur in their greatest degree when the right ventricle is considerably diseased; but in a slight extent I have often met with them when the patient made little or no complaint of the heart, and when the chief sign of its disease was a constant murmur at the apex, indicating regurgitation through the mitral valve. When this defect does not obviously derange the action of the organ, it may still affect other organs through the circulation: continually putting back, at every beat, a little blood into the veins, may produce an accumulative effect in the different organs of the body, and none are so likely to suffer as those largely supplied with venous blood, and have been previously liable to any derangement. There are few cases of diseased heart, especially when this valve is affected, in which the functions of the abdominal viscera are not occasionally disordered; and I repeat, that this disorder is in many cases more prominent than the organic defect from which it has arisen. I cannot tell you how many patients I have had who call themselves, and have been called by their medical attendants bilious, dyspeptic, and nervous, sometimes without a suspicion of a heart complaint, and in whom I have detected lesion of this organ, slight perhaps, but producing these effects by the constancy of its operation. Did time admit, I would give you many examples. In the worst forms of

mitral disease, the conditions of pulmonary congestion and venous obstruction are combined; and then you see the terrible array of symptoms, which has rendered the name of organic disease of the heart so appalling, yet which does not occur in above one in a score of the cases where the structure of the organ is really diseased.

Of the signs of diseases of the *pulmonary semilunar valves* I know nothing from experience. They are so rare that I never met with a case in which I have rightly suspected its existence during life, nor in which I have found, after death, lesions clearly sufficient to produce distinctive signs. The only lesion that I have seen which might possibly have yielded a sign, was atrophy and perforation of the margins of the valves, the loops left by which, perhaps, might catch the current, and give a slight roughness or grating with the first sound. This would probably be audible at the middle of the sternum at its left margin opposite the valve, and perhaps below, over the right ventricle, but not at the top of the sternum or in the carotids; and as the pulmonary artery immediately plunges backwards under the arch of the aorta and into the lungs, it presents no course over which the sound could be traced. Speaking also *à priori*, of *regurgitant* lesions of this valve, if they could be distinguished at all, it would be by a murmur with the second sound heard at the middle of the sternum, and not in the arteries, and unaccompanied by the peculiarities of pulse which attend such a sound when connected with diseased aortic valves.

Of the *right auricular* or *tricuspid valve*, I can speak only as to its *regurgitant* lesions. We have before found that regurgitation through this valve is probably very common; it is chiefly known by pulsating swelling of the jugular veins, and is not commonly attended with any audible murmur. This, I think, may be explained by the laminæ of the valve being so yielding as not to resist the current firmly enough to produce sound, or if a slight sound be produced it is lost backwards. But in a few cases I have heard a deep blowing or grating murmur, distinct from the middle to the bottom of the sternum, in the epigastrium, and at the margin of the ribs close to it, not audible under the left breast, at the top of the sternum, nor in the carotid arteries, and accompanied by pulsating swelling of the jugulars, and signs of dilated hypertrophy of the right ventricle. This murmur I have been induced to ascribe to regurgitation through a tricuspid valve, thicker and more rigid than usual; and in two such cases I have found this condition of the valve after death: in one of them with some constriction of the orifice. Both these cases were combined with great dilatation and hypertrophy, especially affecting the right ventricle and auricle, and the symptoms had been the ordinary ones of extensive organic disease of the heart, affecting the lungs with unusual severity.

The different lesions of the valves and orifices which I have been describing may be variously combined; in which case their diagnosis will be more difficult, for the positive characters of each are seldom so distinctive as to announce them clearly. Murmurs may

be produced in two orifices at once—as by obstructive disease of the aortic and regurgitative disease of the mitral. The former might be recognized by the grating sound in the arteries, but the mitral murmur could not be distinguished from it, unless it were very different in character; as when it is whistling or whiffing, and quite superficial at the apex. On the ground of such differences I have several times founded diagnoses of lesions of both orifices, but I do not lay much stress on them. The physical diagnosis of valvular disease is in some instances very easy, but it is far from being so generally; and in the more complicated cases, accompanied by irregular action, without any definite or well-localized murmur, it often must be too conjectural to be depended on.

We have so little time left for the subject of *diseases of the aorta*, that I must dismiss it in a few words; having little to say that is new, and therefore referring you to the writings of recent authors on the subject, as you will find them epitomized in Dr. Copland's Dictionary.

The topics which we cannot entirely omit, are *dilatation* and *aneurisms* of the aorta, in the diagnosis of which I may, perhaps, be able to assist you. *General dilatation* of the ascending aorta and of its arch frequently accompanies disease of the left ventricle, especially that which affects the aortic valves. It may occur with very little apparent disease in the coats of the artery, and is then more uniform; but more commonly these are much altered, and then the dilatation is more irregular, and with a tendency to form lateral sacs, particularly near the attachment of the aortic valves and below the branching off of the innominate. The alterations of the coats consist in various deposits of opaque matter, differing in consistence and appearance, sometimes softer than the rest of the tissue, and easily separated from the serous coat; in other instances harder and tougher, involving the middle fibrous coat; in either case, there being occasionally osseous scales or concretions in them, and sometimes the whole calibre of the vessels being beset with all these deposits, broken up into the most rugged irregularities. But to do justice to the anatomical characters of these lesions would require a whole lecture; and, rather than describe them imperfectly, I forbear to dwell longer on them.

The aorta thus dilated, and wanting its proper regulating elasticity, receives the contents of the ventricle with more abruptness than usual; this circumstance, with the greater size of the vessel, increases the force and loudness of the pulsations at the upper part of the sternum, above it, and in the carotids. The character and sound of the pulsation will depend on the state of the walls of the artery and of its orifice. If the orifice be not contracted, nor the valves indurated, nor the ventricle much enlarged, nor the walls of the artery very rough or with projecting points, the pulsations will be simply unusually strong, with a double sound; the first sound being exaggerated by the impulse of the blood against the dilated walls of the artery, the second being that of the aortic valves simply transmitted. But if any of these condi-

tions be present, they will constitute a resistance to the moving current of blood, and a murmur, more or less harsh and grating, will be the result, often with a thrill perceptible to the touch. To distinguish whether this murmur is produced in the orifice or in the rugged artery, we must return to the heart, and seek for the signs of obstructive disease of the aortic orifice. If they be not found, and the first sound in the arteries is deep, loud, and harsh, the probability is that it is produced in the artery; and even if there be signs of diseased aortic valves—if the murmur in the arteries be single and much louder, and more harsh than that heard at the middle of the sternum—there is still probably disease of the aorta, which transmits its own sound rather than those of the heart. Dilatation of the aorta above the orifice also increases the murmurs resulting from partial impediments in the orifice; and when the ventricle is also enlarged, the natural orifice becoming *relatively* narrow, may be the seat of a murmur without any real constriction.

Aneurism, partial or saccular dilatation of the ascending or bending portion of the aorta, or of the innominata or the other great arteries, may cause additional signs and symptoms, in consequence of the greater local extent of their tumour. When they are yet small, they may only increase the impulse and sound at the top of the sternum, and perhaps be accompanied with some general symptoms of disordered circulation; but as they increase in size, they press on the adjoining parts, and cause more or less disorder of their functions, and produce many physical signs. Thus their pulsating pressure on the pneumo-gastric nerves may sometimes, at an early stage, disorder the sensations of the lungs and stomach, causing feelings of slight dyspnœa, cough, nausea, or gastrodynia: its increasing bulk may compress—the veins—hence their distension, with lividity, congestion and œdema of the parts from which they come, the head, neck, and upper extremities; the nerves—hence pain, numbness, and paralysis of the arms and hands; the œsophagus—hence difficulty of swallowing; the trachea or its branches—hence bronchitis, severe cough, and dyspnœa; the bones and cartilages—hence caries and absorption, and in case of the spine, perhaps, disturbance of its contents; and lastly, some of the other arteries—whence deficient pulse and circulation in the part to which it leads; occasionally one of the upper extremities. Some or other of these symptoms are generally present in the progress of aortic aneurisms; and their number and intensity may be generally explained by the particular position and size of the tumour.

The physical signs of aneurism of the upper part of the aorta are those of increased pulsation and sound, and, in advanced stages, those of a pulsating tumour presenting itself in various positions. The early increased pulsation may not differ from that of a generally dilated aorta, except, perhaps, that it is perceptible more at one spot above the clavicle, or that its sound is more to one side than towards the middle of the sternum. The sound may be like that of general dilatation, either simply double, like that of the heart, or single,

attended with a grating or whizzing ; the second sound of the heart being inaudible or obscure. The same remark will apply to more advanced aneurisms. They are commonly accompanied by a grating single or double sound at each pulse, but sometimes there is no grating ; both the sounds are loud, and give one the idea that the heart is beating under the top of the sternum. In fact, that aneurisms shall produce a grating, there must be more than simply a sac smoothly communicating with the artery ; the edges of the orifice must be abrupt and projecting, or contracted ; or there must be some roughness or prominence in the artery near it ; or there must be some other vent out of the sac, so that the current may not beat *in* merely, but *whiz through*. If the sac be very elastic, and the orifice small, there may possibly be a jet of blood to-and-fro, in and out of it, at each pulse and interval, causing a double whizzing ; but I have never seen such a case. In most of the cases which I have seen, there has been at the top of the sternum a short grating sound, alternated with a duller, more prolonged sound, which I suppose to arise from pressure or constriction of some of the smaller arterial branches.* To distinguish such sounds from those of diseased aortic valves, you must have recourse to the same expedients as for the diagnosis of general dilatation ; only, in case of aneurism, the sounds may be more local, and not so strongly transmitted to both carotids in the neck. When the aneurism reaches such a size as to form a tumour, perceptible above the sternum or clavicle, or between the upper ribs close to the sternum, the only remaining difficulty is to distinguish it from other tumours : other tumours may produce the same symptoms and signs of pressure, and from the beating of the artery under them they may seem to be pulsating ; nay, by compressing it or one of its branches, they may cause a grating or whizzing at each pulse, very like that of aneurism ; and from the same cause they may disturb the action of the heart. But the pulsation of an aneurism may be generally distinguished by its greater extent, its distension in all directions, and sometimes by its softness ; its sounds are generally much louder ; and the pulsations of the arteries beyond, although occasionally unequal, are generally more free than they would be if a solid tumour were pressing much on their exterior.

Aneurisms of the descending aorta may generally be known by an increased sound of pulsation, generally grating or whizzing, at some point along the left of the dorsal spine. As it advances and forms a tumour forwards, it may produce many other signs and symptoms, by pressing and displacing the lungs and the heart. If it increase backwards, it causes absorption of the ribs and vertebræ, producing paralysis and other symptoms of pressure on the spinal marrow, and presenting its pulsating tumour behind.

*This prolonged sound I now conclude to be seated in the veins : I have heard it in almost every case of substernal tumour that I have met with, aneurismal and other. It is generally a continuous droning note, swelling and rising at each pulse, like the *bruit de diable*, but not so loud. It is a valuable sign of partial venous obstruction.

LECTURE XXX.

On the Prognosis and Treatment of Diseases of the Heart.—On the different Course of Cardiac Lesions.—On the Value of their Signs and Symptoms in directing the Treatment.—Practical Indications common to Diseases of the Heart.—Treatment of functional Disorders; increased Action; defective and irregular Action.—Treatment of Inflammations of the Heart.—Treatment of structural Diseases; Hypertrophy; Dilatation; Diseases of the Valves and Orifices.—Treatment of complex organic Disease of the Heart, and of its Symptoms and Effects.—Remedial Treatment.—Diet and regimen.

THIS, our concluding lecture, is to be devoted to the consideration of the principles and application of the *treatment of disease of the heart*. I am induced to embody my observations on the remedial measures applicable to the different affections which have occupied our attention in the preceding lectures, because I thus avoid repetitions and save time, and because also I shall be better enabled to bring before you the general principles of treatment which are applicable to all diseases of the heart, inasmuch as they all agree in producing more or less disorder in that spring of all the functions, the circulation of the blood.

If we look at the different varieties and degrees of diseases of the heart, even those which so much affect its structure that their nature cannot be a matter of any doubt, we find a wonderful variety in the extent of their effects in the system at large, and consequently in their tendencies and duration. In some cases serious and unequivocal symptoms of heart disease have soon ended in death; yet the lesions found have been comparatively small, and by no means *apparently* proportioned to the severe and fatal character of the affection. In other instances the symptoms have been more equivocal and complicated, less clearly referrible to the heart, being grouped with prominent affections of other organs; yet still, on death ensuing, the chief lesion is found to be in the heart, although even that may not be very extensive. In a third class of cases, and they are very numerous, there may have been the same symptoms as those which occurred in either of the former classes; their severity may have been such that the patients have been thought to be dying under them; yet they have recovered to a valetudinarian state, which has lasted for many months, and even years, sometimes much, sometimes little harassed by similar symptoms; occasionally again brought to death's door by exacerbations of these attacks, yet regaining afterwards health enough to enable them for years to discharge the duties of life; and when at length they die from this disease, or from some other induced or aggravated by it, there is found such an enormous extent of old structural disease, that it is a matter of astonishment to us that life could have been maintained so long and so well.

From witnessing many such cases and such results, I have learned

two things in particular : first, that structural lesions of the heart are attended with immediate danger in proportion more to the quickness of their production or of their increase than to their extent : and secondly, that if acute attacks or intercurrent disorders, which aggravate the permanent lesion, can be prevented or speedily removed, the fatal issue of even extensive organic diseases of the heart may be very commonly delayed for a considerable length of time. Inflammatory affections of the heart may in rare examples destroy life in their acute stage. More frequently they lay the foundation of lesions that proceed slowly and often in a latent manner, until suddenly developed by some new attack or cause of disorder : this may sometimes be accidental, but it is often consequent on the insidious operation of the latent lesion on some of the bodily functions ; and were this discovered in time, it might often be prevented or counteracted. In occasional examples these diseases cause sudden death ; and it is this circumstance chiefly that attaches an awful character to the whole class. But such instances are comparatively rare ; and bear a very small proportion to the cases that we detect either during life or after death. Diseases of the heart are popularly looked on as hopeless cases : and to discover or distinguish them has been pronounced by high authorities in the profession to be the most difficult part of physical diagnosis. Yet, imperfect as our knowledge of this diagnosis is, it is sufficient to prove that diseases of the heart, so far from being always hopeless, not unfrequently exist very long under a mask of delicate health or some vague general disorder ; and by detecting them when they are latent, and by defining them when their general symptoms are equivocal, physical diagnosis has removed from this class of diseases the character of such terrible fatality, and enables us to treat them at a period in which remedial and prophylactic measures may be of much avail.

I have said that diseases of the heart always produce more or less disorder of the circulation ; but the most prominent symptoms of this disorder may be in parts remote from the heart, and may blind us as to their original source. Thus the excited circulation of pericarditis or hypertrophy sometimes manifests itself more in the brain than in the heart ; and the retarded circulation of mitral disease often chiefly declares itself by derangement of the liver. The physical signs lead us back from these symptoms to their first cause, and guide us to shape our practice with a due reference to its character. But are we to disregard these general symptoms or secondary effects ? Certainly not. They often become important parts of the whole disease ; and with regard to the more chronic affections it may be said, that it is in great measure through these secondary effects that the balance of the functions is destroyed, complicated disease produced, the original lesion aggravated in return, and the malady becomes no longer local, but general, affecting first the functions, then the structures of several of the most important organs of the body. We must study disease in all its bearings : in

its earliest history and supposed causes ; for in these we may learn its origin, and be guided aright to investigate its nature. In its physical characters ; for in these we find that of it which is essential and fixed. In its general symptoms and constitutional affects ; for in these we find how and where the local disease is producing in the function or structure of other parts mischief that mainly contributes to injure the vital powers and to aggravate the original evil. And this study is as essential for the proper treatment as for the thorough knowledge of the disease. Thus in a case of pericarditis, if we neglect to inquire into the former history of the subject, we may fail to discover that there was long ago a severe rheumatic affection, followed perhaps by some symptoms of organic lesion which have only been more strongly developed by the present attack : and not knowing this, we may be led to treat the disease with dangerous vigour. Again, if we neglect in the same case carefully to examine the physical signs, we may not discover pericarditis to be present ; but, guided by the prominent general symptoms, mistake it (as it has happened) for phrenitis, or bilious fever, bronchitis influenza, or pleurisy, or gastritis, or some other quite different malady. Under this error the proper treatment may be withheld, and the patient's life lost, or limited by the formation of permanent organic disease. And if we neglect general symptoms, we lose the means of judging of the strength of the bodily powers, of the effects of the disease and of the remedies on them ; and for want of this judgment, we can never rightly proportion or regulate the treatment.

Nor is attention to all the bearings of the disease less necessary in cases of more chronic or structural affections. These, whether their symptoms be distinct or not, if neglected, tend continually to derange the functions of other organs, and gradually to alter their structure, which increases and perpetuates constitutional disease, and reacts on and aggravates the original lesion. The lungs, the liver, the kidneys, the brain, and the mucous and serous membranes, all are liable to functional, and ultimately structural disease, from the disordered circulation caused by an imperfect heart ; and if they are not watched and relieved before they materially suffer, their affections must hasten the failure and decay of the whole frame. The imperfection of the heart itself has also a tendency to increase, from the additional irritation and work thrown on it by its injured mechanism ; and if not placed in circumstances calculated to soothe this irritation, and lighten this labour, its very exertions become more and more hurtful, and soon end in destroying life.

If, on the other hand, we are fully aware of the origin, character, and constitutional tendencies of a disease of the heart, we can often direct its treatment with remarkable success. In the case of inflammation, the application of antiphlogistic remedies with due relation to its seat and character,—and in that of structural disease, the adoption of means proper to lighten the labour of the organ, to allay its excitement, and to prevent the bad consequences resulting from long

continued disorder in parts of the circulation,—are practical applications of this knowledge, the utility of which cannot be doubted. In the more moderate forms of structural disease in particular, in which the cardiac affection is eclipsed and unobserved amongst a variety of secondary symptoms, the knowledge of its existence may be of the greatest utility. The subjects of such disease, according to the effects which it manifests, are called “delicate,” “of a weakly constitution,” “nervous,” “dyspeptic,” “bilious,” or “apoplectic,” and if treated incautiously as such, without regard to the disease of the heart, may be seriously injured in consequence. Thus, for a supposed “general debility,” are freely prescribed tonics and generous diet : for an apparent “nervousness,” or “hypochondriasis,” exciting amusements and active exercise : for a “liver complaint,” reducing courses of mercury and of purgatives : for “blood to the head,” or “tendency to apoplexy,” venesection, cupping, and starvation : and all these measures, by further injuring the balance of the functions, more commonly aggravate than mitigate the original disease. Judicious practitioners, warned by experience, are led to treat these cases with more prudence and success, although still in ignorance of their real nature. Of such cases they will tell you, that although weak, they “do not bear tonics well,” or although full and apoplectic, they “will not stand much reduction.” The practical measures to which they have empirically groped, may be indicated to you by a clear and comprehensive study of the nature of the case ; and in applying and modifying these measures you will always have the advantages and resources which rational and intelligible knowledge will supply above the rules of blind experience.

The treatment of diseases of the heart must, of course, greatly vary, inasmuch as they may be of very opposite characters ; but there is one point of general agreement : the organ is in all cases taxed beyond its force, and whether from excess of irritability, or from defect of power, its task is too much for its well-being. A common indication, therefore, in all cases, is as much as possible to diminish this task, to lighten its labour. The greatest tranquillity of mind and body, without, at the same time, in the more chronic cases incurring the risk of torpor or stagnation ; promoting by means suited to the nature of the case, the capillary circulation ; easing the weight of the general circulating mass by the most favourable postures, varied from time to time to render gravitation an assisting agent ; reducing the quantity of the mass which the heart has to propel, by a sparing use of liquid food ; and at the same time correcting its quality, by cautiously promoting as much as possible the due balance of all the excreting functions ; these are indications applicable to all kinds of cardiac disease, and in many constitute the chief heads of the treatment.

The treatment of *functional disorders* of the heart is a subject of great importance, because it is applicable also to cases of organic disease, which are liable to exacerbations by additional functional disorder. But this treatment must be varied not only according to the

kind of disorder, but often also according to the nature of its cause. Thus *increased action*, arising from general plethora, is best relieved by diminishing the mass of blood by blood-letting, abstinence, and increasing the secretions. That from local plethora, or irregular distribution of blood, if possible, by directing it into its proper channels, and thus restoring the due balance of the circulation; but if the cause of the local congestion be an obstruction to the circulation which cannot be soon removed, or a suppressed secretion that cannot be readily restored, the congestion must be diminished by local or general blood-letting, rather than it should continue to oppress the important functions of the heart and lungs. So, also, when the increased action appears to be from an irritating quality of the blood, as in gouty and rheumatic habits, and in certain fevers, the use of colchicum, alkalies, nitre, and other sedative remedies which act on the secretions, is often found the best mode of relief. Again, inordinate action from extraneous irritation is best reduced by removing, if possible, the irritating cause, whether this be acidity, flatus, or indigestible matter in the stomach, feces or worms in the intestines, congestion or bilious concretions in the liver, an irritation of the uterus, or the like. Or if the cause be only slowly or not at all removable, we may employ means to lessen the sensibility of the nerves and of the heart to its effects, and these are the remedies indicated also where the increased action proceeds chiefly from an excessive irritability in this organ itself; whether this be idopathic, or dependent on a structural lesion. Of these remedies there are two classes; narcotics or sedatives, the action of which is temporary; and tonics, which far less surely but more permanently lessen the irritability of organs, and at the same time augment their power. The sedatives that I have found most generally useful to allay palpitation and inordinate action of the heart, are camphor, hydrocyanic acid, hyoseyamus, belladonna, asafoetida, ether, and opium. The last is generally the most powerful, but its effect on the circulation being irregular, it is apt to disorder the secretions, and occasion subsequent congestions, which may continue to disturb the heart's action. By combining it with mercurial medicines, and ipecacuanha or colchicum, and following its operation by an aperient, these bad consequences may often be prevented. I have found digitalis to exercise far less control over the heart when primarily excited than when its action is quickened by disease of other organs; and as it appears to me, when it does act, to diminish the power of the heart as well as its irritability, I think that its use should be confined to those cases of perpetually increased action, in which a hurtful excess of strength is manifest in the pulse, as well as a frequency of the contractions. Of the other remedies named, I have found all occasionally beneficial, either singly or in combination; but I think that they are more so when given in a few large doses, to break as soon and as completely as possible the habit of morbid action, which, when established, is much more difficult to remove. Hydrocyanic acid must be excepted from this rule, for in large doses it acts like

digitalis, inducing syncope ; but in quantities of two or three minims it is often very effectual in quieting the palpitations caused by a disordered stomach. The tonic medicines generally best calculated to permanently lower the irritability of the heart, or of the nervous system through which it is excited, are those of a metallic nature, such as the subnitrate of bismuth, the sulphate and oxyde of zync, the nitrate of silver, and the sulphate of copper. Whether these act directly on the nerves and heart, or only indirectly by modifying the condition of the alimentary canal, I cannot determine ; but I have known a continued exhibition of each of them, in different instances, decidedly useful in relieving a tendency to nervous palpitation, even where this was combined with some amount of structural disease. Pure country air and cold bathing, or free sponging, with regular gentle exercise, are sometimes more effectual than medicines, in removing palpitation chiefly dependent on a mobility or nervous irritability of the heart. Such a condition may be accompanied by extremes in the action of the heart which is at one time excessive and at another defective, and is then a fit subject for the treatment recommended for weakness or defective action of the heart. On the other hand, if the excessive action be caused, attended, or followed by an inflammatory condition of the organ, evinced by heat of skin, continued pain or soreness, any of the physical signs of endocarditis or pericarditis, or of some new valvular lesion, the treatment should more or less consist of the remedies recommended for these inflammations. Further, it is to be noticed that inordinate action of the heart may produce and leave effects which may require separate treatment, such as pulmonary congestion, and even hemorrhage and inflammation, bronchial spasm and excessive secretion ; nausea and irritability of the stomach ; congestion of the liver and bilious disorder ; severe headache ; swelling of thyroid gland and tonsils, and other results of disturbed circulation.

The treatment of *defective action* of the heart, and of *irregular action* arising out of *weakness*, must also be guided according to its causes and nature. If it be of a transient kind, amounting or approaching to syncope, it may require only temporary measures, which are chiefly the exhibition of diffusible stimuli, such as ammoniacal, alcoholic, and ethereal medicines, or the production of strong impressions on sensitive parts, as by pungent vapours to the nose, dashing cold water on the face and chest, or by drinking a draught of it. Of these measures, none is so effectual as the dash of cold water ; and if combined with warm applications to the extremities. it is the best resource where the fainting fits are so long as to be dangerous. It is not less efficacious in slighter cases ; but it is not then generally eligible, for it is not a favourite remedy. I have known the bare mention of it arouse a lady from an apparent fainting fit, in which the action of the heart had really become very feeble : this shows what power the mind may exert over the heart ; as in instances in which persons have been kept from fainting from loss of blood by strong mental excitement. The defective or irregular

action of the heart induced by flatulent distension of the stomach, or other results of indigestion, is best removed temporarily by ether, or strong carminatives, such as capsicum and the essential oils; and permanently by improving the state of the digestive organs. To guard against the returns of an habitual temporary defection of the heart's action, it is often proper, besides removing as much as possible their causes, to improve the tone and strength of the heart by tonic remedies, and these are also indicated in cases of more permanent or habitual weakness of the organ. Among the medicines generally classed as tonics, none seem to improve the tone of the muscular fibres of the heart so effectually as the preparations of iron, when they are well borne. But it is often very difficult to get a sufficient quantity into the system without disordering the stomach, and causing fever, headache, and other disturbance, which render hurtful the further operation of the medicine. Steel medicines should be begun in very small doses; both preceded and accompanied by occasional mild mercurial or saline aperients, to keep free the secretions, and to prevent any congestion or irritation in the intestinal canal. It is by fulfilling these conditions that weak chalybeate and saline waters sometimes succeed better than steel medicines. The carbonate of iron, too, is sometimes borne from its inertness, for whatever doses be taken, there can be conveyed into the system only the minute quantity that becomes dissolved by acid accidentally present in the alimentary canal; hence its action is very uncertain. The preparations of iron that I have generally found to agree best, are the ammoniated tincture, the tartarized iron, and the iodide, which in solution forms the hydriodate. The last has succeeded in my hands better than any other preparation; especially in chlorotic and scrofulous subjects, on whom it sometimes acts like a charm, restoring quietness and strength to their pulse, colour to their cheeks, vigour to their muscles, and regularity to their secretions and other functions. When iron is not borne, some benefit may be derived from bark, quinine, and other vegetable tonics; and in cases where paroxysms of irregular action occasionally occur in a heart habitually weak, the temporary use of ether, camphor, valerian, and the fetid gums, and courses of the metallic preparations before noticed, of bismuth, zinc, and silver, sometimes prove beneficial. The heart and organs generally may often be much strengthened by the daily use of the shower-bath, or of very free sponging of the chest with cold salt water, or vinegar and water; by removing to a pure bracing air, and using frequent gentle exercise in it, and by great regularity and simplicity of living, with as generous a diet as the circumstances of the case will allow.

The treatment of *inflammations of the heart* is to be conducted on the same principles as that of inflammations in general, with this qualification, that unless in a stage almost too early to be called inflammation, we must not expect to produce by copious blood-letting that complete relief to the symptoms that often follows it in other inflammations. The irritation is here so much localized in the

heart, that were inordinate action entirely reduced, it would leave the organ suffering from the equally hurtful extreme of defective action, from which the circulation would continue to be embarrassed, and the feelings of dyspnœa, faintness, and agitation kept up. Then succeeds the period of reaction, which more readily in the heart than in any other organ might bring back the inflammation, even when the body is suffering from extreme weakness and loss of blood. Whether this be the true view of the case or not, I have been convinced by repeated observations of different kinds of practice, that both pericarditis and endocarditis, and especially if connected with rheumatism, are most safely and effectually treated by *moderate general* blood-lettings, avoiding as much as possible sudden or full impressions on the circulation; and that local bleedings, free and repeated, should in all cases be employed as a measure of at least equal importance. The bowels having been freely opened, calomel and opium, as originally recommended by Dr. Robert Hamilton, should be given until the gums show their effects. This valuable combination seems to be most effectual in large doses, twice or thrice in the day, as some of you have seen it prescribed by Dr. Chambers; and with it, or in the intervals, alkaline salines with ipecacuanha or antimony, and in rheumatic cases colchicum, may be given to allay the fever, to promote the secretions, and general irritation which prevails, before the blood-letting and the mercury have taken their full effect. As the disease becomes more chronic, the fever having abated, but signs of effusion of serum or of lymph remaining, with continued disorder of the heart's action, the local bleeding must be repeated, and the influence of the mercury renewed, from time to time, according to the state of the symptoms. For the febrifuge saline, I generally substitute at this stage an alkaline draught, with two or three grains of the hydriodate of potash, with a view to promote the absorption of the effused lymph; and under these means, combined with blistering or pustular irritation, the symptoms and signs have in some cases gradually disappeared, leaving the action and condition of the heart perfectly normal. In other cases apparent health has been restored, but a murmur remains, a proof of permanent structural lesion, which, however, may be of trifling amount, and, if due care be taken, of no ulterior consequence.

The treatment of *structural diseases* of the heart must be adapted to the nature of the lesion as discovered by the physical signs, and of its constitutional effects, as manifested by the general symptoms. When these lesions are simple, the effects of the several kinds may be quite different, and the plan of treatment must be so likewise. But the lesions are much more commonly combined, and require in the same case mixed measures, which must be varied from time to time, according to the prevalence of local and general conditions of a different character. I will speak first shortly of the measures suited to the more simple lesions, and conclude with remarks on the general treatment of the more common and complicated forms of organic disease of the heart.

The treatment proper for *hypertrophy* of the heart will partake of the character of that suited to increased action, with a sthenic state of the vascular function from which it originates. Medicines, and a regimen calculated to reduce the action of the heart to a low but even standard, are those which are found to give most relief, and to retard and even arrest the progress of the disease. In severe cases, and to relieve a temporary congestion or increased irritation, occasional blood-letting is expedient, but not carried to a great extent, for it then may be succeeded by a hurtful reaction. Evacuations of other kinds are also useful; but the same rule of moderation should be applied to them. The diet should be spare, especially in liquids and stimulating food; but not starving, for this may produce cachectic and nervous disorders, and will injure other organs and functions more than it will reduce the action of the heart. Again, sedative medicines, such as digitalis, hydrocyanic acid, and colchicum, may sometimes be given with good effect; but not by producing sudden or strong impressions on the circulation, for these injure other functions at the time, and their effect is more than neutralized by the subsequent disorder and the irritations of reaction. Tranquillity, too, is highly desirable; but if this amount to a state of sluggish inaction, it will injure the functions of the capillary and seccernent vessels more than it will quiet the action of the heart. It is always to be remembered, that hypertrophy is a disease affecting the slow and constant process of nutrition; and if it is to be arrested or removed at all (and I believe that moderate degrees both of hypertrophy and of dilatation are curable,) it is by means which act constantly and slowly, and not by sudden or strong influences, or by any which continue to disturb the performance of any functions. The cautions against attempting fully or suddenly to reduce the action of an hypertrophied heart are especially applicable to the cases of fat persons, and those of a flabby fibre; for these can ill bear any abridgment of the strength of their circulation; and the embarrassed action which their heart sometimes exhibits, is often from want rather than excess of power.

In the treatment of *dilatation* of the heart, we have generally to use those measures which are suited to the temporary condition of defective or asthenic action from which it originates, and with which it is habitually combined; but its permanency makes it necessary that the more powerful of these resources should be used with discretion, moderation, and reserved rather for occasions of unusual depression or irregularity, than used habitually. But the treatment of dilatation generally should be of as strengthening and nourishing a character as the symptoms will bear; and if depletions or sedatives are necessary, they should be used only so far as to remove the congestions or allay the irritations arising from or accompanying the irregular or defective condition of the circulation. The metallic tonics, especially iron, if it can be made to agree, are often useful in improving the strength of the heart and diminishing its irritability; and its labour may be generally made easier, and its distension less, by

using constant gentle means to keep the mass of the blood as small, and at the same time as rich, as possible. A nourishing diet carefully suited to the capacity of the digestive organs, avoiding that which unduly stimulates, and especially keeping the bulk of liquid at the smallest possible amount; preserving in sufficient activity the superficial circulation and its secretions by warm clothing, friction, gentle exercise, and occasionally, if necessary, by artificial heat, dry or moist; and the cautious preservation of the balance of the excretions by the preceding measures, aided, if need be, by appropriate mild medicines—these are the chief points indicated; and the more completely such a plan can be pursued, the better suited will the blood be in bulk to the strength of the heart, and in quality to improve the tone of its fibres. Occasional attacks of palpitation and asthma, and dropsical effusions, which occur when the preceding measures have been neglected, or have failed to bring relief, will require special treatment; but this must always be applied with the caution necessary with the feeble power of the heart, which can bear no violent remedies, whether of a stimulating or of a reducing kind.

The treatment of *valvular lesions* must be also directed according to the manner in which it disorders the circulation, which varies from constitutional tendencies, as well as from the direct physical effect of the particular kind of lesion. Therefore it is not always but only frequently the case, that diseases of the mitral valve are especially accompanied with venous congestions which require treatment, in the lungs, the liver and bowels, and the head; and that diseases of the aortic valves are attended by more symptoms of arterial excitement throughout the system, requiring occasional moderate depletions and sedative remedies. In fact, scarcely this much can be said of the simple valvular lesions, but only as they commonly occur, combined with enlargement of the respective compartments. We shall therefore pass to the consideration of the general measures useful in the treatment of organic diseases of the heart, in the combined forms in which they generally present themselves.

I have before remarked, that considerable structural lesions of the heart are not incompatible with sufficient health and strength to support for years many of the duties, and to partake in many of the enjoyments of life; and that it is by their sudden development, or by their subsequent aggravation or speedy increase, in consequence of unfavourable circumstances, fresh inflammatory attacks, or of accidental injuries, that they frequently assume their distressing and destructive character. Now, although we can scarcely hope to cure the structural lesion, we may often mitigate its painful effects, and diminish its destructive tendency, by preventing or removing these aggravating attacks, and placing the patient in circumstances the most favourable for the maintenance of the balance of the circulation in its crippled state. The measures for accomplishing these objects may be divided into the *remedial* and the *regiminal*.

The chief *remedial* agents should be reserved as much as possible for times when an aggravation or increase of disease is perceived or may be expected; for if used continually, they may lose their effect, and they may interfere with the resources of nature, that may themselves gradually succeed in adapting the body to the imperfect state of the organ.

The exacerbation of the symptoms of a structural disease of the heart may be the result of mere *nervous* irritation, determined by mental excitement, gastric or intestinal disorder, or some such transitory cause. In this case a few doses of the antispasmodic remedies prescribed for nervous palpitation, may be sufficient, such as ether, camphor, valerian, or the fetid gums. But even these should be combined or followed by an aperient to carry off the effects of the fit of disordered circulation; and in severe attacks of asthma, where there is extensive lesion in the mitral valve, as well as enlargement of the right ventricle, there may be left a congestion in the lungs, which is only to be gradually removed by expectoration, which may be promoted by the decoction of senega, with alkalies, ipecacuanha, and squill, or some other mild expectorant.

It will seldom happen, too, that a severe attack of palpitation, or troubled circulation, affects a diseased heart, without straining and irritating some part of the thickened or altered structure: hence commonly results a local inflammation, which, although slight, may be sufficient to cause a continued aggravation of the symptoms, or repeated returns of attacks of palpitation and asthma. So also sub-acute inflammations are often excited in a diseased heart by exposure to cold, bodily exertion, or mental excitement, the recurrence of rheumatism, or by other inflammatory attacks. It is by removing such inflammation, as well as the local congestions resulting from the unusually disturbed circulation, that local or a small general blood-letting, blistering, and the administration of a few doses of mercury and opium, prove so useful. The judicious application of these remedies, combined as far as it may be indicated with antispasmodics and anodynes, which more promptly allays the excitement and pain of the paroxysms of palpitation and asthma, —and constitutes the chief treatment of the exacerbations of structural diseases of the heart; and the earlier it can be applied, the more mischief it may prevent, and the longer may the imperfect mechanism be made to last. The mode and extent of depletion will be suggested by the circumstances of the case. It should scarcely ever be large. A moderate bleeding from the arm, or cupping from the chest, may be preferred where the signs and symptoms indicate much congestion of the lungs; but where the general weakness or irregular action of the heart renders the loss of blood a questionable measure, considerable benefit with less risk may sometimes be obtained by dry cupping freely employed on the chest and back, or in a less degree by deriving to the extremities by partial hot baths. In most cases, the loss of a small quantity of blood by cupping or leeches to the region of the heart will be sufficiently effectual.

When there is pain or fulness of the right hypochondrium, with an imperfect or disordered secretion of bile, and signs of hæmorrhoidal congestion, the blood-letting may be advantageously practised by leeches to the right side, or in smaller number near the anus. In females the disordered circulation not unfrequently interferes with the regularity of the uterine function; and if in these cases the natural relief cannot be obtained by the means usually employed, it may be imitated and sometimes induced, and the cardiac disease mitigated, by leeches applied to the groins or thighs. With lesions of the mitral valve and right side of the heart, these visceral congestions not unfrequently occur as the accumulated result of slight constant regurgitation, when there may have been no unusual disturbance of the circulation; and in this case, if treated early, they may yield to slighter measures, such as mild mercurial aperients; the occasional exhibition of which, to prevent the recurrence of "bilious attacks," or of hæmorrhoidal annoyances constitutes the best part of the medicinal treatment of many slight cases of disease of the heart, which are often not known to be such.

In the severer forms of structural lesion, the subacute inflammatory exacerbations may often need more than blood-letting; and here a few doses of calomel and Dover's powder, or analogous compounds of mercury with a narcotic and diaphoretic, are often of signal benefit. It is not always necessary to carry them to the extent of affecting the gums. A dose every night, for four or five days, carried off in the mornings by a mild salts and senna draught, will often suffice. But if the increased quickness, sharpness, or irregularity of pulse, palpitation or dyspnœa, with pain or inability to lie down, continue, calomel or blue pill should be given until the system is slightly affected. Under these circumstances, blisters are also often very beneficial; especially where there are signs of effusion into the pericardium or pleura, or considerable bronchial inflammation and secretion. A succession of them is often necessary, or a seton or issue may be substituted for them. In valvular disease, with inflammatory exacerbations referrible to the endocardium, I have found the tartar emetic solution a more effectual counter-irritant than blisters.

When the exacerbations have been in great measure relieved by these measures, their effect may be often kept up by milder means, which do not reduce the strength—such as colchicum, hydriodate and nitrate of potass, and the expressed juice or extract of taraxacum, which may ultimately be combined with, or exchanged for, some mild tonic—such as infusion of calumbo or cascarilla and nitric acid, with a little bitter or aromatic tincture. In these and all other circumstances, the state of the alimentary functions should be carefully watched and its irregularities treated with appropriate remedies, which I need not specify. Nothing is more apt to irritate an injured heart, and to bring on attacks of palpitation or asthma, as disorder of the stomach and bowels.

But the exacerbations of organic diseases of the heart are some-

times the result of weakness and cachexy, rather than of inflammatory action; or if there be a little local mischief of this kind going on, the state of general debility and atony is such that nothing of the antiphlogistic kind beyond a blister, a mustard poultice, or some such slight local treatment, can be ventured on. In such cases, which, if severe, are the most hopeless, stimulants and tonics, as recommended for defective action of the heart, must be used; but from the permanent nature of the lesion, they are soon apt to lose their influence, and must be variously combined and changed, to maintain it as long as possible.

Dropsy, continued or often recurring in its various forms, when unattended with signs of considerable vascular power, is also an unfavourable symptom; for it generally implies either that there is permanent inadequacy of the heart to its task, or that the great secreting organs, the liver and the kidneys, have also become diseased. When dropsical effusions are the result of increased excitement of the heart, or of the supervention on it of an attack of bronchitis, or other occasional disease, they may be only temporary, being the result of an effort of the blood-vessels to diminish the bulk of their contents. But in any case, dropsical effusions may, by their pressure and bulk, interfere with functions, and therefore require special treatment. As long as relief can be obtained by diuretics, where the urine is not permanently albuminous, or by diaphoretics and mild purgatives, where it is, it is better to adhere to these remedies; but when they fail, which they too often do, recourse must be had to the more violent measures—hydragogue purgatives, acupuncture, and paracentesis. Of the diuretics for dropsies from disease of the heart, with considerably increased action, the saline diuretics, nitrate and acetate of potash, with infusion of digitalis or decoction of senega, have appeared to me to answer best. When the heart has less strength and more irregularity of action, particularly if accompanied with albuminous urine, the decoction of the *pyrola umbellata*, with tincture of cantharides and sp. of nitric ether, is more suitable. Squill, with blue pill and a little sulphate of iron, as recommended by Dr. Abercrombie, is also sometimes effectual, but it is very apt to disorder the stomach. In quite asthenic and cachectic cases, I have sometimes seen the tartarized iron prove a safe and effectual diuretic; and I have, in two or three instances, added a grain or two of the iodide of iron to a saline diuretic, with good effect. But the treatment of dropsy is too extensive a subject for our time; and although it do arise so commonly from disease of the heart, even when the kidneys and liver are also in a morbid state, I must refer you to books, as that of Dr. Seymour, and the article on Dropsy in Dr. Copland's Dictionary.

Although the chief medicinal treatment that is needed in structural disease of the heart, is that directed against its secondary effects, or the temporary exacerbations, yet I do not deem it useless sometimes to prescribe for the permanent lesion. It is true that we cannot remove this lesion, but we may make the heart and nerves

less sensitive to its effects. We cannot remove the organic causes of epilepsy, but there can be no doubt that, under the use of nitrate of silver, the operation of this cause is diminished and the fits rendered less frequent. So by the use of the same remedy, or of the subnitrate of bismuth or the oxide of zinc, in organic disease of the heart in persons of a nervous temperament, I have seen a diminution of those occasional exacerbations which constitute the most harassing part of the complaint, and which are in part dependent on an extreme sensibility of the heart and nervous system ; which feel the organic imperfection, and occasion vain and hurtful struggles against it. But this object is more commonly and surely obtained by means of regimen.

I have already said so much of the great importance of preserving a *balance* of the functions in structural diseases of the heart, that you will be prepared to expect that, in the few recommendations which it remains that I should make on the *regimen* proper for them, an equilibrium, or rather an activity of the most even, tranquil kind, will be the principle and aim of the whole plan.

The diet should generally be of as nourishing a quality as will suit the digestive organs, and will not produce plethora or inflammation. It is a great mistake to suppose that diseases of the heart are to be cured by starving. Except when inflammation is actually present, or in case of hypertrophy with continued increased action, or where pulmonary hemorrhage or apoplexy is threatened, the diet should be one of moderation, but not abstemiousness. In most cases real strength in the organs of circulation, and in the muscles generally, is defective, whilst the irritability of the nervous system is undiminished. Now if tone and strength are to be given to the muscular system, and tranquillity to the nervous, by any mode of diet, it is by one of a nutritious character, with a due proportion of good animal food ; and whilst it may be necessary to limit the proportion of the meat, and vary its mixture with less nutritious articles, especially farinaceous, according to the state of the digestive organs and the wants of the system, it is particularly important that in no case should the food, whether solid or liquid, be bulky in quantity. A distended stomach is apt, both mechanically and sympathetically, to excite or disorder the heart's action ; and many are the individuals who suffer from palpitation under such an influence, when at other times they are scarcely sensible of it. The quantity of liquid food should also be limited, for a reason that I have already stated, to keep down the mass of circulating fluid. It is very common for patients with diseased hearts to suffer severely after drinking too freely, although it be only of tea or water. The appetite for excessive quantities of drink is more the result of habit than of real want : by exercising a little self-denial in the first instance, the feelings of thirst gradually abate ; and comfort and health are alike retained by drinking sparingly.

The expediency of avoiding active exertion is so obvious that I

need not dwell on it; but there are particular kinds of exertion, which without appearing to be violent, greatly try the heart's powers. I would instance walking up stairs or up hill, or using even moderate exertion in a constrained posture. Against these the patient must be particularly cautioned. I have known great improvement ensue on a patient removing from a house and country in which he encountered stairs or hills at every turn, to those in which all was nearly on a level. This point is the more important, because moderate exercise is in most cases highly beneficial; and the more of this that can be taken in open healthy air, without inducing fatigue, the better for the strength and regularity of all the functions. This must be done in some cases only on foot; but it is desirable to vary the mode of exercise, and to those accustomed to ride, no motion is more suitable or salutary than the gentle paces of an easy horse. Where riding is not borne, carriage exercise is the only other resource; and it may well be combined with walking, but, when taken alone, it is inferior to either of the others. Travelling by easy stages about a pleasant country is sometimes beneficial in many ways—by the amount of moderate exercise, the change of air, and the mental amusement, which it affords. In the intervals of exercise, which constitute the greater part of the day, it is very essential that the body should really be at rest; and by easy horizontal or inclined postures, on sofas, couches, or other contrivances, that the labour of the heart be aided as much as possible in propelling the blood to all parts. When the capacity for exercise fails, it is proper to assist the defective circulation, particularly in the extremities, by daily friction. In all cases this may prove salutary, by promoting the function of the extreme vessels and the skin. With the same object it is highly important to have a sufficiency of woollen clothing; and if the skin is habitually dry, its condition may be improved by warm salt water baths. In a relaxed state of the surface, sponging freely with tepid or cold salt water or vinegar, followed by friction, proves equally salutary.

Moral and mental quietude is most desirable, but here again not inactivity. According to the natural dispositions, various degrees of mental occupation and social intercourse are necessary; and so strongly is the action, and thence the structure, of the heart influenced by metaphysical causes, that there is no morbid circumstance more pernicious than violent mental passions, and no medicine, in chronic cases, so salutary as the calm enjoyment of agreeable social relations, and of amusing intellectual pursuits. We speak *practically* not less than *poetically*, when we place these passions in the human heart; and as we hear of many a whole heart rent and broken with grief and trouble, or burst with unbridled passion, we may well judge what havoc these feelings may cause in a heart already unsound. It is not often ours to "minister to a mind diseased, or cleanse the stuffed bosom of that perilous stuff that weighs upon the heart;" but we may often enlist the patient's understanding and

well-grounded fears on our own side, when we counsel him sobriety in his sorrow and calmness in his care: on us may devolve the privilege and responsibility (and in no case does the conscientious physician, with his best exertions, more need divine help to direct his judgment aright) of advising and deciding with regard to important changes in the relations and occupations of his future life.

THE END.

